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SYDNEY, SATURDAY, MARCH 5, 1955

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HOSPITAL RECORDS.¹

By J. C. FULTON,

Chief Executive Officer and Medical Superintendent,
Royal Alexandra Hospital for Children, Sydney.

THE title of this talk is "Hospital Records", a title which embraces all the records of the hospital—that is, medical, X-ray films, detailed pathological investigations and all the non-medical departmental records—but I propose to limit the discussion to records usually maintained by the medical records department. Time will not allow for any consideration of X-ray films.

Why Do We Have Medical Records?

Why do we have medical records? The answer to this is that they contribute towards the better care of the patient. Their value lies in the following: (i) treatment of the present illness and the future illnesses of the patient, (ii) legal defence, (iii) education, (iv) medical research. It is hardly necessary to point out nowadays that the use of instruments of precision results in such a volume of data expressed in exact terms that it is utterly impossible for the physician to retain and to keep properly separated

in his mind the infinite detail regarding each of the large number of patients he must attend. This applies to the present illness, and even more so to the problem of future illnesses. Without a record of past illness a great deal of repetition becomes necessary, entailing unnecessary expense and waste of the time of an already busy physician.

MacEachern has made the following statement:

In an attempt to provide a remedy, the hospital has assumed the burden of keeping medical records for the physicians of the Medical Staff, with the result that in a large majority of cases a wrong attitude has unconsciously developed, which is harmful to the patient as well as to the hospital and the physician. The physician often feels that he is conferring a favour on the hospital when he contributes to the medical records. While the medical record undoubtedly is of some value to the Hospital, its greatest value is to the physician himself, and if there is any favour conferred, the Hospital confers it in assuming the responsibility for maintaining an adequate records system. As a matter of common courtesy, if for no other reason, the physician should be willing to see that the data is accurate and complete.

There is no doubt that the keeping of medical records creates a better scientific approach to the treatment of the patient.

It is becoming increasingly common for actions to be brought against physicians and hospitals arising out of the treatment of patients. In the vast majority of cases

¹ Read at a meeting of the New South Wales Branch of the British Medical Association on November 25, 1954.

the action is unjust, and adequate defence relies on the preservation of accurate medical records of the case.

The educational value of medical records applies both to resident medical officers and to the more experienced physician. It is hardly necessary to mention the value of case studies for members of the resident medical staff, while sometimes the importance to the experienced physician of the study of cases and groups of cases is overlooked. Without recorded case studies a discussion of cases is not based on exact data, and it degenerates into abstract theorizing which is apt to lead nowhere. However, the greatest educational value of medical records lies in the impossibility of evading errors. No person likes to acknowledge mistakes, and consequently evasion is an inherent weakness of human nature. Yet errors are the best teachers, particularly when they are brought to light and the cause is sought.

From the point of view of research, the hospitals are a vast laboratory for observing the course of disease and the effect of new methods and new treatments—their advantages, disadvantages and limitations. In many hospitals one of the greatest obstacles to this type of research is the incompleteness of medical records.

What is the Medical Record?

We can now ask ourselves, what is the medical record? Mrs. Edna Huffman defines it as follows:

It is a clear, concise and accurate history of the patient's life and illness written from the medical point of view. To be complete, it must contain sufficient data written in sequence of events to justify the diagnosis and warrant the treatment and end results.

Mrs. Huffman further states that "to ascertain whether it will meet these criteria, the medical record must be analysed quantitatively by the medical records librarian, and qualitatively by the physician". In somewhat greater detail MacEachern defines a complete medical record as being one which includes the following:

Identification data; complaint; personal and family history; history of present illness; physical examination; special examinations, such as consultations, clinical, laboratory, X-ray, and other examinations; provisional or working diagnosis; medical or surgical treatment; gross and microscopic pathological findings; progress notes; final diagnosis; condition on discharge; and in case of death, autopsy findings.

The Making and Keeping of Records.

Next, one may ask how records are made and how they are kept. The various persons taking part in the compiling of records in the hospital are the nurses, the special investigators, and the doctors. However, there is only one person responsible in the hospital for the medical record—that is the attending physician. He is the one who fully knows the patient, knows all the facts, and is competent to arrive at conclusions. He is the only one who can properly certify to the accuracy and completeness of the record as it applies to the individual patient.

The actual method of making the record is to write the details out by hand, to use recording equipment where possible, and to make use of medical secretaries and medical stenographers in certain places in the hospital. In many of our hospitals the major part of the record is written by the junior resident medical officer.

Ideally the attending physician signs the record at the termination of treatment, indicating that he is satisfied that the document is accurate and complete and agrees with the written diagnosis.

There is room for argument as to whether nurses' notes form part of the medical record. My own experience over the past few years in a number of unjust claims is that nurses' notes have been essential in the rebuttal of the charges; I refer to the graphic charts maintained by the nursing staff and also to the night and day ward reports. Anything which tends towards safer keeping of these documents is most desirable legally.

Throughout the world the two major practical problems which arise in medical records departments are as follows: (i) the difficulty of having full and accurate data recorded, (ii) adequate storage facilities for the accumulating mass of records. I shall deal with the second one later on.

Attempts to rectify the first difficulty by securing the cooperation of physicians have been only partially successful. One important step in this direction has been the creation of a medical records committee—a committee of the medical staff. Its duty may be stated simply as seeing that accurate and complete medical records are secured for every patient treated in hospital. Three to five members make a satisfactory committee. The role of the medical records librarian here is to make a quantitative analysis of the medical record and see that all available parts are in the record before it is submitted to the committee. However, she is not qualified to pass judgement on the quality of the content and should not attempt to do so. This is the function which is delegated by the medical staff to the medical records committee.

It is obviously impossible for the medical records committee in a large hospital to inspect every record before it is passed as satisfactory. This may be overcome by the committee inspecting samples of the medical records; for example, every tenth or every twentieth record may be submitted for their perusal. The committee may appoint individual members to carry out this work, especially when specialist knowledge in assessing the quality of the history is necessary.

In the case of unsatisfactory records, the action of the committee through its secretary would be to write a polite letter to the medical attendant concerned, pointing out the deficiencies. Should this fail to bring about a satisfactory result, a second but sterner letter would be sent to the member concerned. Failure to secure action by this means would then result in the committee reporting to the medical staff. Should the medical staff fail to bring about a satisfactory result, then the medical staff may report the member to the governing body. Whether or not the medical records committee is going to be successful will depend entirely on the support it gets from the governing body.

A second important duty of the medical records committee is to influence the whole medical staff into an appreciation of the value of the medical record and of their personal responsibilities.

Before leaving this aspect of medical staff responsibility, I should like to say that I have seen little reference in text-books to the attending physician's moral responsibility to his patient to see that a proper medical record is kept. Whether it is a private patient or a public patient, I believe that the attending physician, when he undertakes the medical care of a patient, also undertakes as part of his contract to see that a proper record is made of the illness. This is because the patient will not receive the best possible care in his present illness unless adequate records are kept, nor will he be in a position for future illnesses to be treated by a medical attendant having a full knowledge of past investigations, diagnosis and treatments.

Our next consideration is as to how the medical record is kept. If one is in the fortunate position of designing a new hospital, location of the medical records department becomes one of the most important and interesting aspects of design. This location should be such that the department serves equally well the in-patient section of the hospital and the out-patient department. This location makes it possible for the records of a patient, whether out-patient or in-patient, to be united in a single record. This is known as a unit record system. It means that, from whatever part of the hospital a call is made for the record, the record received has all details of that patient's treatments in the hospital, whether they have been in the out-patient department, in special clinics or in the in-patient wards. There is no doubt that this is the most satisfactory system of keeping a patient's medical record. When separate records are kept for out-patient attendances and for in-patient admissions, difficulties arise,

much unnecessary work has to be repeated and the patient is often involved in unnecessary expense. If one has to make a decision as to whether the location of the medical records department should be closer to the in-patient section or to the out-patient department, probably it is wiser to site it nearer the out-patient department, because more files are called for by the out-patient than by the in-patient department. The combining of the in-patient and out-patient records in one is known as the unit record system, and the filing of these records in one single location in the hospital is known as the centralized unit record system.

The principal requirement of a medical records department is that records should be available at all times. If a medical records department fails in this regard, the principal reason for its existence vanishes, because the record must be available when required, so that effective treatment of the patient can be carried out. It is here that many hospitals and hospital superintendents have failed to give as much help as they should to the medical records department staff.

A second important consideration is the means of transporting records between their points of usage and the medical records department. The use of skilled labour in the form of nurses as messengers is completely unjustified, and the three principal methods are as follows: (i) messenger service, (ii) pneumatic tube transport, (iii) chain belt system. In the United States of America the pneumatic tube system has been giving satisfactory service for a number of years and seems to be standard practice in new hospitals. An essential part of a centralized unit record system is immediate availability of the record anywhere in the hospital; this is greatly facilitated by a pneumatic tube system for receiving and dispatching records.

When the record is completed and received into the medical record department for filing, very complex and exacting work is required by the staff of the department. This staff consists of medical record officers, keepers or librarians *plus* medical record clerks.

In the modern hospital the medical records librarian has become a most important member of the hospital team. She is a highly trained and ethical person who has definite responsibilities but not with regard to the quality of the medical data.

A medical records librarian is a person trained to assemble and analyse the component parts of the medical record; to determine whether it will justify the diagnosis and warrant the treatment and end results before filing; to keep it readily available for use in future illness of the patient, in medico-legal need, or for research and study; and to collect but not evaluate the medical statistics needed by the hospital.

Here in New South Wales one of our most pressing problems is to bring about the training of more medical record librarians.

During a recent visit to the United States and Canada I noted the following staffs of medical records departments: Los Angeles Children's Hospital (300 beds, 8000 in-patients and 100,000 out-patient attendances), 18 personnel; Toronto Hospital for Sick Children (740 beds), 25 full-time personnel; University of Chicago Clinics (750 beds, 200,000 out-patient attendances), 31 personnel; Society of New York Hospital (1411 beds), 60 to 70 personnel. The medical records department of this last hospital, the New York Hospital, works round the clock, uses centralized unit records with open shelf filing by the terminal digit method and has pneumatic tube transportation.

Time does not allow for any detailed reference to medical record room procedures, but mention will be made of systems commonly used and of some recent developments.

Security and Preservation of Medical Records.

The following rules serve as a guide to the security and preservation of medical records.

1. The record remains the property of the hospital and should be preserved for the benefit of the patient, for research and teaching purposes and for protection of the medical attendant and of the hospital against any unjust claims or criticism.

2. The hospital may involve itself in legal proceedings at the suit of a patient if it discloses, without the patient's consent, personal data obtained confidentially during the course of investigation and treatment. Such consent should be obtained in writing.

3. The contents of the record should not be disclosed in legal proceedings unless the patient or his legal guardian consents. Otherwise the record can be brought into the court only upon subpoena.

4. In the release of information, if the information is to be used impersonally—for example, for research and publication—it is not necessary for the patient to consent; but the permission of the medical attendant should be obtained as a matter of courtesy. Should patients request information from their own medical records, the information should be made available to their medical attendants. In certain special circumstances, however, it may be desirable to make the information available to the patient.

5. There is no specific time limit set by law for the preservation of records. It is advisable that they be kept during the lifetime of the Statutes of Limitations. In general, these provide, in the case of adults of sound mind, that all actions based on civil wrongs, such as negligence, are to be brought within six years from the date on which the cause of action accrued. However, in the case of infants, time does not begin to run against the infant until the infant attains the age of twenty-one years. Similarly, time does not begin to run against a person of unsound mind when the right of action first accrues. Many authorities consider that records may be destroyed after twenty-five years.

6. There are special provisions relating to the disclosure of information in relation to venereal disease. Before disclosing the name of a patient who has suffered from venereal disease, or before giving any information, a medical practitioner should be familiar with Sections 4 (3) and 19 of the *Venereal Diseases Act, 1918*.

Medical records relating to venereal disease should be produced in a court by a medical practitioner only, who should secure the direction (and protection) of the court before disclosing the information.

Numbering, Filing, Storage, Classifying and Indexing.

Once adequate and accurate medical records are being secured, it is necessary to file them so that any required record may be found promptly; this is the criterion by which the records department's efficiency is judged.

Filing records alphabetically has now given way to the numerical method, in which the patient's record is given a number and then filed according to various systems, such as the serial numbering method or the unit numbering method. The latter is the one being adopted by most hospitals. In this the record is given a number on the patient's first admission to hospital, and that number is retained on all subsequent admissions to any department of the hospital.

There are various methods of storing records which will not be mentioned here, except to say that the inadequacy of storage space is a serious problem in nearly all hospitals, whether new or old. This problem is accentuated when in-patient, out-patient, special department and nurses' records are combined in the centralized unit record system.

It is the amount of storage space available and its accessibility to the medical records staff which determine how long a hospital can keep its records. Various methods of photographing and microfilming records have been tried with a view to reducing their bulk, but each method has had unsatisfactory features. When microfilming of records onto 16-millimetre roll film has been practised, difficulties arise with the use of the film viewer and magnifier, and

also with the inconvenience of using rolls of film each containing many individual records.

Lately a method of cutting roll microfilm into short lengths, which are inserted into small cards or "Cellophane" holders, has been adopted in the United States of America. A small card (for example, eight inches by five inches) then holds the record of an individual patient, and the number of cards can be increased as the record grows. A difficulty with this method is still the high cost of viewers when a large number are required for wards and clinics.

It is suggested that a combination of retaining original records as long as possible, say for fifteen years (this being their most active period), and then microfilming them by the "Cardex" method will meet the storage problem. I did hear of some small hospitals which were microfilming their records immediately and mounting them in small cards.

Classification and Terminology.

For comparison of records and compiling of statistics it is obvious that hospitals and countries must speak a common disease terminology before any accurate conclusions can be drawn.

The most difficult matter of policy for a hospital to decide at present is the terminology to be adopted for the description of diseases, conditions and operations. There have been many local nomenclatures and now there are two main national nomenclatures for use in hospitals—the Nomenclature of the Royal College of Physicians in Britain and the Standard Nomenclature of Diseases and Operations of the American Medical Association in the United States of America. On an international level there is the International Statistical Classification of Diseases, Injuries and Causes of Death of the World Health Organization.

It has been held by many that the International Classification, being designed mainly for statistical study of large groups or whole countries, is not suitable for the clinical detail which the medical staff of a hospital studies in a relatively small number of individuals. Accordingly a great many hospitals have adopted the United States Standard Nomenclature, which provides in great detail a compromise classification based on topography and on aetiology in the case of diseases, and on topography and procedure in the case of operations.

The coding system used in the United States Standard Nomenclature is complex, but it does give the means of pulling out records to satisfy fine clinical detail.

It is somewhat disturbing to find that in the past two years the Columbia-Presbyterian Medical Centre in New York has changed from the Standard Nomenclature to the International Classification. This was a dramatic decision, if it is realized that the Presbyterian Hospital was one of the experimental hospitals in 1932 which first tried and then developed the Standard Nomenclature. Now, after twenty years of using it, the hospital has changed to the International Classification. The reasons given were that the coding, which is irksome, was not being done by the medical attendant and was being left too much to lay coding clerks, who made wrong interpretations. Thus, when records for a piece of research were needed there could be missing material. Another difficulty was the maintenance of an experienced staff of lay coders. The Presbyterian Hospital will not yet give its opinion of the International Classification, but believes that the research man who asks for material now, because of the broader groupings of the International Classification, gets what he asks for, even though he may have to do a little discarding of some unwanted material in the records pulled.

Some hospitals in the United States of America are continuing to use the Standard Nomenclature, but are coding material on a diagnostic index according to both the Standard Nomenclature and the International Classification.

The Nomenclature of the Royal College of Physicians does not yet seem to have been tested against its value for cross-indexing.

I think we shall get some more conclusive opinions when the next International Congress on Medical Records is held in 1956.

Indices.

Lastly, the medical records librarian maintains a name index, diagnostic index, operations index and any special index which will assist in locating records for a special piece of research.

Summary.

1. The basic responsibility of a medical records department is to be able to produce a complete and coherent medical history concerning any and every hospital patient as and when it is required.

2. The personnel involved in making this possible are the medical attendants and nurses, the medical records librarian and staff, and the hospital administrator.

3. The physical means most suited to carrying this out is the centralized unit-record system, together with mechanical aids, such as the pneumatic tube system and some degree of records photography.

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COMPLEMENT FIXING ANTIBODIES FOR THE PSITTACOSIS-LYMPHOGRANULOMA GROUP OF VIRUSES AMONG NORMAL PEOPLE IN SOUTH AUSTRALIA.

By D. SURREY DANE.¹

From the Institute of Medical and Veterinary Science, Adelaide.

THE complement fixation test for the psittacosis-lymphogranuloma group of viruses is used widely as a diagnostic procedure in cases of possible virus pneumonia, and also in suspected cases of *lymphogranuloma venereum*. A group-reactive antigen is employed, and consequently the test shows only whether infection with one of the viruses of this group has occurred; it does not show which particular virus may have been responsible for an infection. The interpretation of results depends to some extent on a knowledge of the antibodies present in the normal population. Because little was known about the occurrence of these antibodies among normal people in South Australia, a serological survey was made. The results of the survey, and certain investigations which followed, are reported in this paper and their significance is discussed.

Materials and Methods.

Serum.

Serum from 607 people was examined. Of these people, 401 lived in or near the town of Waikerie on the Murray River, and the remaining 206 lived in Adelaide. The Waikerie sera were collected from normal individuals of varying ages during September, 1953. The majority of the people from whom the blood was taken were living in the town of Waikerie itself, but some came from the neighbouring towns of Morgan and Blanchetown, and a few from the

¹ Assisted by a grant from the National Health and Medical Research Council.

surrounding countryside. In Adelaide, serum was collected from a number of different sources during August, 1953. Blood was collected from most of the children aged under five years, in hospital, where they had been admitted for a variety of reasons. The majority of older children and adults came from the suburb of Norwood. All were in good health at the time when blood was collected from them. Serum was stored at -15°C . and inactivated at 56°C . for thirty minutes before being tested.

Complement Fixation Test.

The psittacosis-lymphogranuloma group antigen used in the complement fixation test was prepared from chick embryo yolk-sac infected with the virus of enzootic abortion of ewes (Stamp *et alii*, 1950). A detailed description of the preparation and qualities of this antigen is given elsewhere (Dane, 1955). It gives similar results to the type of heated psittacosis virus antigen described by Bedson and his colleagues (1949), and now used extensively for routine diagnostic investigation. Calcium magnesium saline (Mayer *et alii*, 1946) was used for making all dilutions, and 0.2 millilitre quantities were used for each reagent in the test. Two minimal hæmolytic doses of pooled guinea-pig complement were used. The highest dilution of antigen giving the highest titre with a known reacting human serum was determined by a checker-board titration. Twice this amount of antigen was used in the test. Dilutions of unknown sera were incubated in a water bath for one hour at 37°C . with complement and antigen, and then 3% sheep's red cells sensitized with five minimum hæmolytic doses of hæmolysin were added. After a further thirty minutes at 37°C ., unlysed cells were deposited by centrifugation and the results were read. The titre of a serum was taken as the highest dilution in which there was 50% or more fixation. Serum controls and yolk-sac antigen controls were included in each test. The normal yolk-sac antigen was prepared in a similar manner to the virus antigen, but used at twice the strength. Complement was check-titrated in each test. Each serum was tested in doubling dilutions from one in five to one in 40. Any serum giving complete fixation at one in 40 was titrated out in a subsequent test; and sera giving doubtful reactions were also tested again. None of the sera tested showed any fixation of complement with the control antigen, and only a few sera were slightly anti-complementary.

Results.

Waikerie.

The percentage of sera from people in different age groups having titres of one in five or greater is shown in Figure 1. Of the children aged under five years, 6%, and of the children in the five to nine years age group, 8%, had positive titres. The incidence rose to 20% in the ten to fourteen years age group, and to 26% in the fifteen to nineteen years age group. It remained at about this level in the older age groups. The positive titres found varied between one in five and one in 160. Twenty-eight sera gave positive results at one in five dilutions, and the number of sera giving positive results at each higher dilution became progressively fewer until only two gave positive results at one in 160 (see Table I). There were about the same percentage of positive reactions among males as among females, and from the data available it did not appear that the people having positive titres fell into any definite social or occupational groups.

Adelaide.

The percentage of sera from people in different age groups having positive titres of one in five or greater is shown in Figure 2. The distribution of antibodies was similar to that found at Waikerie in the two younger age groups; but among older people there was a slightly higher percentage of positive reactors. The positive titres found varied between one in five and one in 160, with progressively fewer occurring in the higher dilutions (see Table II). Antibodies were equally common in males and females. Among the positive reactors there was one obvious group. Blood had been taken from fourteen boys at a primary

school; they were aged between ten and twelve years. Ten of these boys gave a positive response to the complement fixation test, which was a considerably greater number than would have been expected to judge from the results obtained with other children of this age tested in Adelaide and at Waikerie.

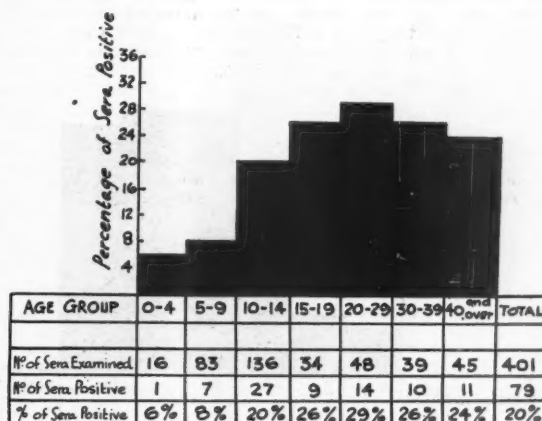


FIGURE 1.

The percentage of Waikerie sera from subjects in different age groups having titres of one in five or greater.

Further Investigations at Waikerie.

In March, 1954, six months after the original blood samples had been taken at Waikerie, blood was again taken from 34 people who had given positive results to the complement fixation test. They were also questioned about their past health and about contact with birds and animals. At this stage of the investigations the question of which

TABLE I.
Positive Titres in Waikerie Sera.

Reciprocal of Serum Titre.	Number of Sera Giving Positive Findings.
5	28
10	24
20	15
40	6
80	4
160	2
Total	79

virus or viruses of the psittacosis-lymphogranuloma group were responsible for the positive reactions had to be considered. *Lymphogranuloma venereum* is exceedingly rare in this State and was unlikely to have been responsible for any of the positive reactions. The possibility of this infection was therefore ignored, except that people were asked whether they had ever been abroad. Inquiries were made about past attacks of pneumonia, as these might possibly have been caused by a psittacosis virus¹ or by a pneumonitis virus of the type described by Eaton, Beck and Pearson (1941). Infection with the viruses of trachoma and inclusion blennorrhoea may give rise to low titre psittacosis-lymphogranuloma group antibodies (Rake *et alii*, 1942). Though neither condition is now common in the areas covered by the survey, questions were asked

¹ The term "psittacosis virus" is used in this paper to include all bird viruses of the psittacosis-ornithosis sub-group.

about past conjunctivitis. Non-specific urethritis, which may sometimes be caused by the virus of inclusion blennorrhoea (Lindner, 1910), is rarely seen at Waikerie (Miller, personal communication), and no questions were asked about it.

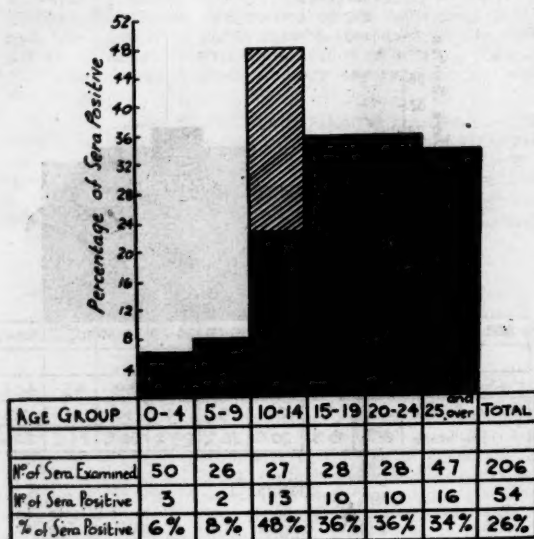


FIGURE II.

The percentage of Adelaide sera from subjects in different age groups having titres of one in five or greater. The hatched area in the ten to fourteen years age group includes the results obtained at the primary school mentioned in the text.

The fact that so many people had been shown to have antibodies suggested that some minor respiratory infection might be caused by a virus of this group, and therefore people were asked about recent attacks of influenza, sore throats and colds. It was realized that memories are usually short and inaccurate for these types of illness.

TABLE II.
Positive Titres in Adelaide Sera.

Reciprocal of Serum Titre.	Number of Sera Giving Positive Findings.
5	18
10	18
20	9
40	5
80	2
160	2
Total	54

People who have contact with birds often give a positive response to the complement fixation test without giving a history of any past illness resembling psittacosis. For this reason questions were asked about contact with psittacine and other birds. A brief general past medical history was taken, and contact with other known positive reactors was asked about when possible.

The results of testing the paired sera from September, 1953, and March, 1954, are shown in Table III. In only one case was any significant variation in titre found. A boy (W381), aged ten years, had a titre of one in 160, which fell during the six months' interval to one in 10. He gave

no history of any recent illness. The answers given to some of the questions which the people were asked are also shown in Table III. Contact with birds was fairly general, as might have been expected, because many families in Waikerie keep domestic fowls, and psittacine birds are common household pets. A history of past pneumonia was given by 10 of the 34 people, but in the majority of cases this illness had occurred many years previously. Six people gave histories of a conjunctivitis which might possibly have been due to the virus of trachoma or inclusion

TABLE III.
Results from 34 Persons at Waikerie with Positive Titres.

Serial Number.	Age (Years) and Sex.	Complement Fixation Test Result.		Contact with Birds.		Past Pneumonia.
		September, 1953.	March, 1954.	Psittacine.	Others.	
W2	M., 45	20	20	—	—	Aged 28 and 42.
W12	M., 41	5	10	—	+	Aged 34.
W13	M., 38	10	10	—	+	Aged 25.
W15	M., 29	5	5	—	+	—
W20	F., 17	10	5	—	+	—
W25	F., 32	20	10	—	+	As an infant.
W30	M., 39	20	10	+	+	—
W40	F., 3	20	N.T. ¹	—	+	Aged 3 months.
W82	M., 30	20	40	+	+	—
W86	M., 14	160	80	—	+	—
W88	F., 24	20	40	+	+	—
W116	M., 13	10	20	—	—	—
W117	M., 11	5	N.T.	—	—	—
W119	F., 43	20	20	—	+	—
W123	F., 8	20	N.T.	—	—	Aged 3.
W154	F., 26	20	10	—	+	—
W177	M., 30	5	10	—	+	As an infant.
W182	M., 44	20	40	+	+	Aged 5.
W183	M., 40	80	80	—	—	—
W186	M., 20	10	5	+	+	—
W187	M., 50	80	80	+	+	—
W206	F., 12	10	5	+	+	—
W232	F., 11	20	20	—	+	—
W248	M., 13	5	5	+	+	—
W252	F., 39	40	40	—	—	—
W273	F., 22	40	40	—	+	—
W320	F., 13	80	40	—	—	—
W325	F., 13	40	40	—	+	—
W350	M., 15	20	20	—	—	Aged 13.
W361	F., 16	10	20	—	—	—
W362	F., 23	10	10	—	—	Aged 12.
W381	M., 10	160	10	—	+	—
W403	F., 27	5	5	—	+	—
W405	F., 27	40	20	—	+	—

¹ N.T., not tested.

blennorrhoea. None of the other questions asked gave any clue as to the type of infection which might have been responsible for the positive results to the complement fixation test.

Further Investigations in Adelaide.

It was possible to interview 12 out of the 14 boys from the primary school in April, 1954, nine months after their original blood test. A further sample of blood was taken from ten of them, and they were asked the same questions about their past health and contact with birds as had been asked at Waikerie. The results of testing their paired sera are given in Table IV. Two boys had significant rising titres. One boy (N118) had a titre of one in five which rose to one in 40, and another (N120), who had had a titre of less than one in five on the first occasion, had a titre of one in 640 nine months later. None of the boys had histories of past pneumonia or conjunctivitis. As a group, their contact with birds was not in any way out of the ordinary, and they had no common contact with any birds. In answer to questions about illness in the twelve months preceding August 23, 1953, when blood was taken from them for the first time, only three definitely remembered being ill. One boy, N119, had had pharyngitis in April, 1953; another, N120, had had laryngitis "shortly before" blood was taken from him for the first time; and the third, N127, had had pharyngitis in March, 1953. The two boys with rising titres over the nine months' interval did not remember being ill during that period, and the school

records showed that they had not missed any days at school. The records of absenteeism at the school failed to show evidence of epidemics in 1953 which appeared significant. One explanation of the findings at this school could be that in 1953 there was an outbreak of mild respiratory disease caused by a virus of the psittacosis-lymphogranuloma group. The evidence for this is too slight to do more than suggest possible future lines of research.

TABLE IV.
Results from 14 Boys at an Adelaide Primary School.

Serial Number.	Age. (Years.)	Complement Fixation Test Result.*		Contact with Birds.	
		August, 1953.	April, 1954.	Psittacine.	Others.
N115	11	5 ¹	5	+	+
N116	11	<5	<5	—	—
N117	12	160	N.T. ²	Not questioned.	—
N118	10	5	40	—	+
N119	12	10	20	—	+
N120	11	<5	640	+	+
N121	11	5	N.T.	—	—
N122	12	40	N.T.	—	—
N123	10	<5	N.T.	Not questioned.	—
N124	11	5	10	—	+
N125	10	20	20	+	+
N126	10	<5	<5	—	+
N127	12	80	40	—	+
N128	12	20	20	+	+

* Reciprocal of serum titre.

² N.T., not tested.

Discussion.

The results of the survey showed that complement fixing antibodies to viruses of the psittacosis-lymphogranuloma group are common amongst the general population of South Australia. Somewhat similar results have been obtained in surveys in Holland, Denmark and the United States. Dekking in Holland (quoted by Meyer and Eddie, 1951) found that 10% of persons not clinically infected with a member of the psittacosis-lymphogranuloma group of viruses had a titre of one in eight, and 5% had a titre of one in 16. In Denmark, Flyvstrup and his associates (1950) found that 15% of 308 sera from various sources gave a positive response to the complement fixation test at one in five dilution or greater. Most of the strong reactions were found in patients with atypical respiratory infections, especially pneumonia. In the United States, Beeson and Miller (1944) found that 33% of coloured people in Atlanta attending hospital for a variety of reasons gave a positive response to the complement fixation test with lymphogranuloma venereum antigen to a titre of one in five or greater. A similar group of white people had an incidence of 10% positive reactors. Whilst these workers interpreted these findings as being primarily due to lymphogranuloma venereum infection, they were unable to assess the part played by other viruses of the group.

The results of the present survey pose two main problems. In the first place, do the positive titres in fact represent definite evidence of past or present infection with a virus of the psittacosis-lymphogranuloma group? Secondly, if they do, what particular virus or viruses are responsible? The following points are either in favour of or compatible with the reactions being specific. (a) The tests were controlled by the use of a normal yolk-sac antigen of at least twice the strength of the virus antigen, and in no case was there any sign of fixation with the normal antigen. (b) There is a definite relationship between age and percentage of positive reactors. (c) The positive titres found in normal individuals were generally maintained at a fairly constant level over six or nine months. (d) Titres of one in 40 or more are generally accepted as evidence of past or present infection with some virus of this group. If these higher titres are to be accepted, it seems reasonable also to accept the lower titres found in the survey, which occur only in the sort of proportion that might be expected (see Tables I and II).

On these grounds, and in the absence of any evidence suggesting that the reactions were non-specific, it was concluded that the positive titres represented evidence of past or present infection with a virus of the psittacosis-lymphogranuloma group.

The second problem—which virus or viruses were responsible for the infection—is unsolved. Lymphogranuloma venereum can be ruled out as a likely cause of more than a very occasional positive reaction because of its rarity. Trachoma and inclusion blennorrhoea are uncommon in the areas covered by the survey, and as only a few of the people with positive titres who were questioned gave any history suggestive of either disease, these viruses were probably not the cause of many positive reactions. Recently it has been suggested that sheep and cattle viruses of the psittacosis-lymphogranuloma group may at times infect man (Enright and Sadler, 1954; Gerloff and Lackman, 1954). Few of those questioned at Waikerie or in Adelaide had had any direct contact with these animals, and unless humans are infected from cow's milk it is improbable that these viruses were of importance.

Psittacosis is seldom diagnosed in South Australia. During the twelve months from June, 1953, to May, 1954, sera from 134 patients with pneumonia or pyrexia of unknown origin were examined at this institute, and in only one case could psittacosis be diagnosed serologically with some certainty (Beech, 1954). The serological diagnosis of psittacosis is made difficult by the use of antibiotics, which appear to affect the production of antibody (Sigel *et alii*, 1953; French *et alii*, 1954) and for this reason some cases may have been missed. However, the large number of positive reactors in the survey, and the absence of any history of pneumonia in most of those people with positive titres who were questioned, makes it unlikely that clinically recognizable attacks of psittacosis were responsible for many of the positive results to the complement fixation test. On the other hand, mild or inapparent infection with viruses of avian origin could possibly have been the cause of most of the positive reactions. It is known that aviary owners, pigeon breeders and pet shop employees frequently have positive titres without giving a history of any illness resembling psittacosis (Meyer and Eddie, 1948). It is also known that in this State many of the birds commonly in contact with man have a high rate of infection with psittacosis, and thus are a potential source of infection for man (Beech and Miles, 1953; Dane and Beech, 1954). Few people, even if they do not normally come into contact with birds, can avoid doing so occasionally either directly or indirectly. Whilst the theory of widespread, mild or inapparent infection with bird viruses is hard to prove or disprove, it is at least a possible explanation for the results obtained in the survey.

Another explanation is that there are viruses of the psittacosis-lymphogranuloma group which are primarily human parasites, and generally cause no more than a mild respiratory tract infection. The investigations at an Adelaide primary school, which are reported in this paper, lend some support to this theory.

Summary.

1. Serum from 607 people living in South Australia was examined for complement fixation antibodies to the psittacosis-lymphogranuloma group of viruses. Titres of one in five or greater were demonstrated in 22% of the sera. Antibodies were more common in adults than in children.

2. Evidence is presented that the presence of low titre antibodies is more likely to be due to past or present infection with a virus of the psittacosis-lymphogranuloma group than to non-specific causes.

3. The problem of what virus or viruses are responsible for the majority of the infections is discussed.

Acknowledgements.

My thanks are due to Dr. J. A. R. Miles for his help and advice, to Dr. R. L. Miller for his help in the investigations at Waikerie, to the headmasters and staff of the

various schools at which blood was taken from children during the survey for their cooperation, and to Miss Fay Makin for her technical assistance. I would also like to record my thanks to the many people at Waikerie and in Adelaide who volunteered to have their blood tested in the survey and in the follow-up investigations.

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Method.

Twenty-four students living in their family homes and 16 students living in institutions where the food is cooked in bulk were examined by two observers independently with particular reference to tongue grade. This was estimated in accordance with the scale given by Bolton, the mean of the two observations being recorded. The grades are as follows: grade 0, a normal tongue covered by normal papillae; grade I, a swollen, oedematous tongue, as shown by the indentations produced at the edges by the teeth; grade II, a tongue with smooth edges and tip, the papillae at these sites becoming worn down and atrophic; grade III, a tongue with hypertrophic papillae towards its centre, fusion of which produces early fissuring and "cracking" on the upper surface, the edges being smooth; grade IV, a mammillated tongue, most of the papillae having atrophied; grade V, a smooth, shiny, completely atrophic tongue.

TABLE I.
"Home Student" Series.^{1,2}

Number.	T.	Hb.	SA.	TP.	DTP.	FCP.	MCh.	MEg.
I	0	100	4.5	7.5	89	62	10	43
II	1-25	100	5.8	7.4	95	74	30	37
III	1-0	106	4.5	6.8	73	56	11	37
IV	1-25	112	4.8	6.8	102	70	33	37
V	1-25	90	4.6	6.8	77	54	20	28
VI	1-25	85	4.6	7.6	67	43	12	25
VII	1-5	98	4.8	7.2	85	68	17	46
VIII	0-75	103	4.1	7.5	66	44	18	23
IX	0-25	106	4.8	7.2	93	79	15	61
X	0	104	4.8	7.2	85	70	11	56
XI	1-25	103	4.6	7.2	78	52	14	35
XII	0	104	4.3	6.8	53	42	4	31
XIII	1-25	94	4.6	7.2	67	51	24	23
XIV	1-25	105	4.5	7.5	95	71	12	54
XV	1-0	100	4.5	6.9	108	84	24	56
XVI	1-25	110	4.5	6.9	206	112	32	73
XVII	0-25	108	4.6	7.2	81	53	20	24
XVIII	0	103	3.7	7.2	114	70	24	37
XIX	1-5	115	4.5	7.3	97	56	19	32
XX	0-75	102	4.1	7.2	—	—	—	—
XXI	0-75	110	4.5	7.3	—	—	—	—
XXII	1-0	91	4.5	6.4	—	—	—	—
XXIII	1-75	95	4.5	7.0	—	—	—	—
XXIV	0	106	4.6	7.1	—	—	—	—
XXV	—	—	—	—	62	54	24	27

¹ T, tongue grade; Hb, haemoglobin percentage (100% = 14.8 grammes per centum); SA, serum albumin content (grammes per centum); TP, total protein content (grammes per centum); DTP, dietary total protein intake (grammes per day); FCP, first class protein (grammes per day); MCh, milk and cheese (protein) (grammes per day); MEg, meat and eggs (protein) (grammes per day).

² Six students were unable to arrange suitable times to complete the investigation.

³ Numbers V, VI, XII, XIII, XXIII and XXV were females.

All the students were clinically healthy, and specimens of urine were biochemically normal and macroscopically clear.

The "institution students" were then given a diet rich in protein (and other essential factors—Appendix I) while they returned to their homes on a two-weeks vacation. Finally, they were reassessed on their return.

Serum albumin and total protein estimations were performed on the "home students" and on the "institution students" before and after the test diet, the methods employed being those in routine use in this hospital—the former by the method of Folin and Wu (1919) after precipitation by 42% sodium sulphite solution (Campbell and Hanna, 1937) with the use of a Hellige photoelectric colorimeter, and the latter by the copper sulphate specific gravity method (Phillips *et alii*, 1945). The serum globulin content was not specifically estimated. Blood samples, taken after the clinical assessment, were sent to the laboratory without indication as to the student group from which they came. The blood was taken from an arm vein with the subject seated, no tourniquet being left in position for more than one minute. The students presented about an hour after breakfast or lunch, similar numbers in each group coming at each of these times, and repeated estimations were made at the same hour as the first one. The students realized that alterations in fluid balance would

TONGUE APPEARANCE AND SERUM ALBUMIN LEVEL IN THE ASSESSMENT OF NUTRITIONAL STATUS, WITH REFERENCE TO THE EFFECTS OF A PROTEIN-RICH DIET.

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A RELATIONSHIP has been demonstrated between the sequence of changes in the tongue leading to complete atrophy and the dietary intake of meat and eggs (Bolton, 1955). Bremner, using Bolton's classification, showed a difference in tongue grade between students living at home and students living in colleges (Bremner, 1952). The present investigation was undertaken to confirm the latter observation, and to extend it by estimation of serum albumin level and by examination of the effects of a protein-rich diet. By these means information has been obtained as to the value of tongue grade and serum albumin level as indices of group nutritional status in respect to protein.

affect the results, and they tried to avoid these; if necessary, the estimations were postponed for a day. A portion of the same specimen was sent for haemoglobin estimation (by means of a Gray-Wedge photometer), although capillary blood, taken at the same time, was used in the case of two or three of the "home students".

In addition, detailed dietary histories were taken from the students living at home by a dietitian working independently.

Results.

The results for the "home student" series are set out in Table I, in which the dietary protein intake is also shown, and for the "institution students" before and after the test diet in Table II; the results for both series are summarized in Table III.

The mean tongue grade of the "home students" (0.85) is significantly lower than that of the "institution students" before the test diet (1.39), but does not differ significantly

TABLE II.
"Institution Student" Series.¹

No. ²	Before Diet.				After Diet.			
	T.	Hb%.	SA.	TP.	T.	Hb%.	SA.	TP.
I	1.75	100	4.2	7.5	0.5	86	4.8	6.2
II	1.5	99	3.9	6.8	1.0	91	4.9	7.5
III	1.25	104	4.2	6.9	1.0	91	5.0	7.2
IV	1.0	102	4.2	7.0	0.5	90	4.2	7.3
V	1.25	97	5.0	7.5	1.0	99	4.3	6.9
VI	1.25	99	3.8	7.3	1.0	100	4.9	6.9
VII	1.5	98	3.7	7.0	0.5	97	5.2	7.2
VIII	1.5	113	3.5	6.7	1.0	102	4.6	6.9
IX	1.0	100	3.4	6.9	1.0	102	3.2	6.8
X	2.0	103	3.9	6.9	0.5	100	5.0	7.4
XI	1.5	106	3.9	7.0	1.5	94	4.6	7.3
XII	1.5	111	4.3	7.0	1.0	97	5.0	7.4
XIII	1.0	111	3.5	6.7	1.0	100	4.3	7.0
XIV	1.5	113	4.3	6.9	1.0	110	4.2	7.0
XV	1.5	115	4.9	7.3	1.0	105	4.9	7.2
XVI	1.25	112	4.5	7.5	1.0	107	4.3	7.5

¹ Hb, haemoglobin percentage (100% = 14.8 grammes per centum); T, tongue grade; SA, serum albumin content (grammes per centum); TP, total protein content (grammes per centum).

² Numbers I to V inclusive were females.

from that of the "institution students" after the test diet (0.91). It is considered that this result cannot be attributed entirely to bias on the part of the observers. Some of the students noticed a change themselves, papillae in some instances developing where there had been none before.

The serum albumin content is highly significantly greater in the "home students" than in the "institution students" (4.57 and 4.07 grammes per centum respectively). After the test diet the level in the latter group rose to 4.59 grammes per centum, which does not differ significantly from that of the "home students". Particular interest is given to this rise of 0.5 gramme per centum in the serum albumin level by the presence of a concomitant significant fall in the haemoglobin value of 7%. The mean total protein values are statistically identical in all three series, and the haemoglobin level of the "home students" does not differ significantly from either of the "institution student" groups. The daily protein intake for individual students living at home is shown in Table I, and the mean values are set out in Table IV. The average daily intake by college students of "animal protein" (approximately equivalent to the "first class protein" of the present paper) has been estimated by Bremner at about 46 grammes. Questioning after the vacation showed that the "institution students" had adhered to the diet for at least ten of the sixteen possible days, and in all cases the protein intake had been increased above the pre-vacation level.

Thus, in a series of volunteer students, subdivided on a basis of place of abode, the students living at home fared significantly better in respect of tongue grade and serum albumin level than students living in institutions. This difference was no longer demonstrable after the latter group had been given a protein-rich diet for two weeks.

Tongue Grade and Serum Albumin Level as Indices of Group Nutritional Status.

The demonstration of significant differences in tongue grade and serum albumin level between two groups of clinically healthy medical students suggests that these two estimations are relatively sensitive indices of group nutritional status, according to the available evidence, in respect of protein. A more detailed analysis of the data was therefore undertaken to define their value in this regard.

TABLE III.
Mean Values for All Groups.¹

Group.	Tongue Grade.	Hb.	SA.	TP.
"Home students" (Group H); 24	0.85	102.0	4.57	7.13
"Institution students" before test diet (Group IA); 16	1.39	105.2	4.07	7.06
"Institution students" after test diet (Group IB); 16	0.91	98.2	4.59	7.11

¹ Hb, haemoglobin value (grammes per centum); SA, serum albumin content (grammes per centum); TP, serum total protein content (grammes per centum).

Initially, the measurements used in the discriminant analysis were the tongue grade (T), serum albumin content (A), total protein content (TP) and haemoglobin value (Hb). The best linear combinations of these measurements to discriminate between the groups H ("home students") and IB ("institution students" before test diet) and the groups IB and IA ("institution students" after the test diet) are given respectively by the following formulæ:

$$x = 0.22 A - 0.021 TP - 0.0053 Hb - 0.25 T$$

and

$$y = 0.21 A - 0.038 TP - 0.010 Hb - 0.22 T.$$

The mean values of x for groups H and IB differ significantly from each other; similarly the mean values for y for

TABLE IV.
Mean Dietary Protein Intake of "Home Students" (Grammes per Day).

Students.	Total Protein.	First Class Protein.	Milk and Cheese.	Meat and Eggs.
Male students (15)	98	68	19	43
Female students (5)	65	49	17	27
Male and female students (20)	90	63	19	39

groups IB and IA also differ significantly from each other (both at the 0.1% point). No discriminant function was calculated to distinguish between groups H and IA.

If an individual is to be classified into one of the two major categories, H or IB (the "treated" group, IA, may be excluded from further consideration for this purpose), it will be necessary to calculate from his measurements the characteristic

$$z = 0.22 A - 0.030 TP - 0.079 Hb - 0.24 T.$$

If z lies below -0.35, he is classified as belonging to the group IB, and if z lies above this value, as belonging to group H (or IA).

The estimate of the probability of misclassification is found to be approximately 0.45, which is high when compared with the value of 0.50 that would apply if classification was made by the random tossing of a coin. This result arises because the differences between the centres of the groups are small relative to the spread of the groups. Its effect is to make the method of little use in the classification of individuals into either of the two groups studied.

Nevertheless, the significant differences between the means provide evidence that the samples come from populations the centres of which are genuinely different. It must be remembered that this difference does not imply the existence of a demonstrable clinical difference between individual members of the groups, although the groups themselves appear to be clinically distinct.

If haemoglobin value and serum total protein content are not estimated, the appropriate linear combination is

$$z^* = 0.24 (A - 1.06 T).$$

The cut-off point here is 0.74; values of z^* lying below this point are allotted to the category IB, and above this point to group H (or IA—again, discrimination between groups IA and H has not been considered). The classification of individuals by this method has the same drawbacks as in the four-variate method above. The estimated probability of misclassification for groups of 94 individuals is approximately 0.125. This does not differ significantly from the estimated probability of misclassification for groups given in the four-variate method, so that there is no evidence in the data to show that the inclusion of haemoglobin value and serum total protein content in the formula for z improves the discriminating power. However, it is possible that study of a larger sample might supply such evidence.

Apart from their interest in relation to the theoretical significance of the equation for z , these findings may well have wide practical applications. Thus a group of not less than 94 individuals, known to be homogeneous, could be classified into one or other of the two major categories by substituting the group means for the two or four variates in the equation for z or z^* respectively, the estimated probability of misclassification being 0.01 or less. Further work may be expected to extend the number of categories, and to define these more accurately in terms of protein intake. The available evidence indicates that such a classification would reflect the dietary protein intake of the group studied, and as such it would constitute a valuable technique for use in nutritional surveys.

Discussion.

The observation that the serum albumin level is relatively sensitive to changes in dietary protein intake supports the clinical impression that, when the level is low owing to malnutrition, it may be raised to normal or near normal values more quickly than is generally believed, provided that extensive liver damage is not present. Largely as the result of Himsworth's work, the comparative failure of a protein-rich diet to influence serum albumin content in the presence of cirrhosis of the liver has been appreciated for many years. Weech (1939) observed the rapid regeneration of albumin in dogs in the absence of liver damage, and he also noted the value of beef and egg white in the diet in producing this effect.

The same observation also raises the difficult question of the determination of normal standards. As far as we have been able to ascertain from a survey of the literature over the past twenty years, no series of "normal" values for serum albumin content in human beings has been reported in which normality is defined partly in terms of protein intake, or in which the values obtained are related to the protein intake of the group studied. The more complex relationship of haemoglobin level to dietary protein requires further study over a longer period than was possible in the present investigation; but there is evidence that similar precautions may be justifiable in the defining of normal haemoglobin levels.

The association of a rise in serum albumin content and a fall in haemoglobin content after the test diet suggests a relationship between serum albumin level and blood volume; in animal experiments Weech and others (Weech, 1939) have demonstrated a fall in plasma volume as the serum albumin level falls. Some results in accord with this hypothesis are mentioned by Adams (1954) in regard to treated patients with kwashiorkor. However, in severe grades of protein deficiency, as in kwashiorkor and famine oedema; both haemoconcentration and haemodilution have been reported as occurring, and it may be that an increase

in circulating blood volume follows a stage of haemoconcentration.

It may be pointed out that the constancy of the mean total protein values in all three groups of students, despite considerable variations in the serum albumin fraction, supports the now generally accepted view that the estimation of serum total protein content alone has little clinical significance, and virtually none if the result is in the normal range.

Summary.

1. In a series of 24 students living at home, the mean grade of tongue change was significantly less, and the mean serum albumin content significantly higher, than in a series of 16 students living in institutions.

2. After two weeks on a protein-rich diet, the "institution students" showed significant improvement in mean tongue grade and serum albumin level, the values becoming identical with those for the "home student" series.

3. Statistical analysis indicates that tongue grade and serum albumin content, or more particularly the appropriate mathematical combination of these, are relatively sensitive indices of group nutritional status in respect to protein, a finding which has wide practical application in studies of human nutrition.

4. The clinical significance of these observations is briefly discussed, with particular reference to the influence of dietary protein intake on blood volume, and on serum albumin and haemoglobin concentrations.

Acknowledgements.

We are indebted to the Department of Statistics, University of Melbourne, and in particular to Miss A. G. Doig and Dr. F. E. Binet, for the detailed statistical analysis required, and to Miss Betty Laby for her assistance with the computation. Our thanks are also due to Mrs. J. Ross, of the Chief Dietitian's staff, for taking the dietetic histories; to Miss June Crawford and Mr. C. Francis, of the Clinical Biochemistry and Pathology Departments of the hospital, for performing the various estimations; and to Miss Beryl Splatt, Dr. D. C. Cowling, Sister B. London and the students themselves for their cooperation in the investigation. The active interest and assistance throughout this project of Dr. J. H. Bolton, Honorary Physician to In-patients, is gratefully acknowledged.

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Appendix.

In a typed sheet issued to all the "institution students" and intended to be taken home for reference, it was stressed that "in view of the short-term nature of the experiment . . . a consistently high and continuous protein intake must be achieved", and "an ideal, well-balanced diet must be aimed at for the same reason". The recommended diet was as follows:

Protein must be taken at every meal with intermediate feeds of milk, and milk or other protein for supper. Milk may be flavoured as desired and skim milk taken if weight gain feared. Minimal requirements: 2 eggs, 8 oz. meat, 2 oz. cheese, 2-3 pints of milk, per day; liver or kidneys once or twice per week; steak or beef once or twice per week; 2 tablespoonfuls "Weetbix" and 1-1 teaspoon "Marmite" or "Vegemite" per day; one good helping of green leafy vegetables per day; 1 orange or 1 grapefruit or juice thereof daily; salad foods (lettuce, carrot, tomato) at least 3 times per week; fat and carbohydrate to taste.

A LABORATORY STUDY OF THE POLIOMYELITIS VIRUSES IN VICTORIA INCORPORATING THE TYPE II EPIDEMIC OF 1952.

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WITH the advent of the new tissue culture techniques for isolation and typing of poliomyelitis viruses and for the estimation of serum antibodies to these viruses, the virus types responsible for poliomyelitis epidemics in Australia may now be readily identified.

Information regarding epidemic and endemic virus strains is available for Victoria since 1949. Tissue culture techniques were not in use in Melbourne during the 1949 epidemic, but appropriate faecal and spinal cord specimens were collected and Type I virus was isolated in America from these specimens, through the courtesy of Dr. Karl Meyer, of the George Williams Hooper Foundation, University of California, San Francisco, United States of America. Bazeley and Thayer (1954a) have reported the first results obtained with tissue culture in Melbourne, and have indicated that Type I strains were again epidemic in 1951, whereas Type II strains made their appearance in late 1952.

The present communication gives laboratory data for the 1952-1953 epidemic, for which Type II virus was responsible, and for the current 1953-1954 epidemic, which is caused by Type I. Some of the clinical aspects of the 1952-1953 Type II epidemic will be the subject of a further communication (Forbes, 1954).

Materials and Methods.

An attempt was made to select groups of patients with paralytic and non-paralytic infections at suitable intervals throughout poliomyelitis epidemics, in order to determine the epidemic strains involved. Faecal specimens and serum samples were obtained at Fairfield Hospital. The faecal specimens were stored in a deep freeze unit at -20°C . until needed for examination.

All laboratory work for this study was carried out at the Commonwealth Serum Laboratories, Melbourne. Technical procedures used in tissue culture have already been described (Bazeley and Thayer, 1954b). The early specimens were tested in monkey testicular tissue with "C" medium as nutrient. Trypsinized monkey kidney tissue (Bazeley, 1953, personal communication) and medium 199 (Morgan, Morton and Parker, 1950) were then adopted and provided a more sensitive cell system for virus isolation. Faecal and spinal cord specimens which failed to yield virus in monkey testicular "piece tissue" were re-tested in trypsinized kidney tissue, with considerable improvement in the Type II virus isolation rate.

Results.

A total of 97 faecal specimens, from Fairfield Hospital patients suffering from paralytic and non-paralytic polio-

myelitis, were examined over the period from September, 1952, to March, 1954, inclusive. Virus strains were isolated from 47 (48%) of these patients, the proportion of isolations being somewhat, but not significantly, higher in paralytic infections (30 strains from 56 specimens) than in non-paralytic infections (17 strains from 41 specimens). The virus types isolated, when grouped into four-month periods, are shown in Table I.

All patients from whom virus strains were isolated, and from whom serum samples were obtained, had antibody to the appropriate serological type of virus. More than half these patients showed heterologous as well as homologous type antibody. Such heterologous antibody was taken to indicate prior infection. Antibody titres varied from one in four to greater than one in 128. A rising titre was demonstrable in some patients when the first serum sample was obtained within three or four days from the onset of the illness.

There were 60 patients from whom no virus was isolated or no faecal specimen was obtained. The antibody levels in these patients are shown in Table II.

It will be seen that 32 of these 60 patients had antibody to one type only. If the clinical diagnosis of poliomyelitis is accepted, then it is reasonable to assume that the infecting type may be inferred in those who have a single antibody. All such patients had evidence of meningeal involvement, with increased numbers of cells in the cerebro-spinal fluid. Although comparatively few sera were examined during 1953 and 1954, and most of the 1954 sera had multiple antibodies, the type diagnosis by serology accords well with the virus types found by isolation.

If the types of infection inferred from serological tests are added to the types determined by isolation of the infecting strain, we have a clearer idea of the change in epidemic type which occurred from 1952-1953 to 1953-1954. The combined figures are listed in Table III, together with the monthly admissions to hospital of paralytic poliomyelitis patients over this period.

Discussion.

The 1952-1953 epidemic began in the early spring and reached its peak in December, 1952. Over this period 26 of the 29 infections in which the infecting type was determined were caused by Type II virus. In the next four months, from mid-summer to autumn in 1953, the poliomyelitis incidence was declining steadily, and the proportion of infections caused by Type II had declined sharply. It is unfortunate that more patients were not investigated over this period.

The Type I epidemic is interesting because it would appear to have started about mid-summer (February, 1953), whilst the Type II epidemic was current, but in sharp decline. The poliomyelitis incidence was negligible during the winter months, but in early spring the epidemic recommenced with Type I infections playing a dominant role and some Type II infections still occurring. This epidemic reached its peak in autumn (March, 1954), at which time 14 of the 15 infections, in which the causal virus was known, were due to Type I.

TABLE I.
Virus Isolations from Fairfield Hospital Patients for the Period from September, 1952, to March, 1954.

Date.	Paralytic Poliomyelitis.			Non-Paralytic Poliomyelitis.			Total.		
	Type I.	Type II.	Type III.	Type I.	Type II.	Type III.	Type I.	Type II.	Type III.
1952: September to December ..	0	6	1	0	7	0	0	13	1
1953: January to April ..	1	1	0	4	4	1	5	5	1
May to August ..	0	0	0	0	0	0	0	0	0
September to December ..	7	1	1	0	0	0	7	1	1
1954: January to March ..	12	0	0	0	1	0	12	1	0

¹ Only three specimens were examined during this period of time.

TABLE II.
Serum Neutralization Tests on Patients Not Included in Table I.

Date.	Paralytic Poliomyelitis.				Non-Paralytic Poliomyelitis.				Total.			
	One Antibody Only.			Multiple Antibodies.	One Antibody Only.			Multiple Antibodies. ¹	One Antibody Only.			Multiple Antibodies.
	Type I.	Type II.	Type III.		Type I.	Type II.	Type III.		Type I.	Type II.	Type III.	
1952: September to December	1	7	0	4	1	6	0	6	2	13	0	10
1953: January to April	0	2	2	1	0	0	0	1	0	2	2	2
May to August	2	0	0	1	0	0	0	0	2	0	0	1
September to December	2	1	1	3	2	2	1	3	4	3	2	6
1954: January to March	2	0	0	4	0	0	0	5	2	0	0	9

¹ A fourfold or greater rise in titre to Type III virus was observed in one specimen of serum in December, 1952, and in two sera in December, 1953.

Type III virus was responsible for sporadic infections throughout the period under study, and similar sporadic incidence has been noted in earlier Victorian work (Bazeley and Thayer, 1954a). It seems probable that Type III infections in Victoria are subclinical in a majority of instances, because though there has been no evidence as yet of a Type III epidemic, yet in serological age group studies (Thayer, to be published) the proportion of persons having Type III antibody is as high as the proportion having Type I and Type II antibody.

TABLE III.
Virus Types Responsible for Infections in Fairfield Hospital from September, 1952, to March, 1954, Inclusive (from Tables I and II).

Date.	Type I.	Type II.	Type III.	Monthly Admissions of Paralyzed Patients.
1952: September } October } November } December }	2	26	1	7 8 33 35 ¹
1953: January.. } February } March .. } April .. } May .. } June .. } July .. } August .. } September } October } November } December }	5 2 11	7 0 4	3 0 3	25 ¹ 23 17 8 1 1 0 1 2 3 9 4
1954: January.. } February } March .. }	14	1	0	12 20 28

¹ Type I infections were not detected during December, 1952, or January, 1953.

All virus strains isolated have proved to be typical of the poliomyelitis viruses, with one exception, which has not been included in this series. This virus, in monkey kidney tissue, produces cytopathogenic changes which are not neutralized by type-specific poliomyelitis antisera, and on present evidence appears to belong to the group of "orphan" viruses described by Melnick (1954).

Summary.

Laboratory studies in Victoria have given the following indications:

1. The 1952-1953 poliomyelitis epidemic was caused by Type II virus.
2. The 1953-1954 epidemic began whilst the Type II epidemic was still current, lapsed in the winter, finally

reached a peak in the following autumn, and was caused by Type I virus.

3. Type III infections occurred sporadically over both epidemic periods.

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ACCIDENTAL INJURIES IN PRE-SCHOOL CHILDREN: I. A GENERAL SURVEY.

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In Australia deaths from accidents are now the principal cause of death of children aged from one to fourteen years. A recent study of the mortality from accidents in childhood (Clements, 1952) showed that the pre-school period was the most important, because of the high accident rates in these ages, and because these are the formative years during which a child's awareness of accident hazards should be developed. However, mortality figures derived from official statistics do not provide information about the many factors which may be associated with the occurrence of an accident, or about types of accident. Without information of this kind it is difficult to plan adequate preventive measures. Case studies of the children treated for accidents either as in-patients or as out-patients at hospitals are one way of building up knowledge about associated factors. This method is expensive, since it generally means the employment of special investigational staff, and the number of children who can be studied in a year is relatively small. Furthermore, an inquiry of this kind is limited to the children who have accidents, and it is impossible to decide whether apparently associated factors are different from those in a non-accident group.

With the object of collecting information about the non-fatal accidents sustained by pre-school children, it was decided to make a survey of a relatively large number of

¹ Endowed by the Commonwealth Department of Health.

TABLE I.
Accidents in Pre-School Children.

Type of Accident.	Total Accidents at All Ages.		Number of Accidents at Ages (Years).											
			Under One Year.		One Year.		Two Years.		Three Years.		Four Years.		Five Years.	
	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.
As a passenger in a motor vehicle ..	297	221	12	7	20	21	64	48	94	62	69	50	38	33
Motor vehicle accident as a pedestrian ..	194	125	1	5	7	4	20	12	41	38	64	36	61	30
Burns and scalds ..	862	711	92	62	207	151	235	189	172	150	81	106	75	53
Falls ..	2536	1882	85	75	236	217	576	439	622	498	581	395	386	258
Poison swallowed ..	702	142	8	7	36	43	111	54	522	25	22	7	3	6
Accident by machinery ..	136	87	2	0	13	8	31	22	39	27	32	19	19	11
Near drowning ..	135	102	3	3	14	15	34	34	32	27	32	14	20	9
Injury by an animal ..	120	111	5	0	11	10	32	38	53	27	25	23	14	13
Foreign body in an orifice ..	115	110	10	9	14	16	23	21	30	39	22	15	16	10
Crushed by falling object ..	133	149	7	4	9	26	40	28	36	38	20	33	21	20
Struck by flying object ..	245	193	5	7	10	13	41	33	47	50	76	56	66	34
Cut by sharp or piercing instrument ..	384	214	11	2	15	14	57	32	98	61	114	61	89	44
Total ..			241	181	642	538	1264	950	1296	1042	1138	815	808	521
Total number exposed at ages ..			12,131	11,653	11,727	11,481	10,690	10,583	10,690	10,583	10,690	10,583	10,690	10,583
Percentage of children who had an accident in this year of age ..			1.98	1.55	5.47	4.68	11.82	8.97	12.12	9.84	10.64	7.7	7.55	4.92

children by the *questionnaire* method. This procedure is always open to a number of objections: it depends upon the cooperation of a large number of people; it calls for a high degree of application to the problem by the parents; and it must be a retrospective study with all the faults of these investigations. Despite these objections, it was considered that some useful information would emerge which might help in planning educational campaigns.

The Collection of the Data.

After a small pilot study to test the form of the *questionnaire*, cards were designed and printed. Each card had spaces for the name, age, sex and position in family of the child, and for the number of children in the family. The main recording device was in the form of a chess board of 54 squares, arranged in six columns of nine rows. A column was provided for each year of age from "under 1 year of age" to five years of age inclusive, and one row was allocated to each of the types of accident. The last row stated: "The child did not have an accident in this year of age." Each square was numbered from 1 to 54. The card contained the two following requests addressed to the parents or guardian of the child: (a) to put a cross (x) in the square corresponding to the accident in the appropriate year of age or a cross (x) in the squares in the last row when no accident occurred in a particular

year of age; (b) on the back of the card, to write a brief account of how each accident occurred, setting out the nature of the injury, if the child was treated by a doctor, at a hospital or by a chemist, and if the accident had left any after-effects on the child. Each description was to be identified by quoting the number of the appropriate square.

No attempt was made to collect information about any fatal accidents to other children in the family. However, a number of parents did give details of fatal accidents to another child of the family. This information has not been used because of its selected nature.

The cards were distributed to all kindergartens in Australia affiliated with the State Kindergarten Unions, and by courtesy of the Minister of Education to a selected group of infants' departments in State schools in New South Wales. A reasonable distribution to schools of all sizes in each zone of the State was ensured by the help of the Research and Guidance Branch of the Department of Education. The teachers were asked to arrange for the parents of children aged three to eight years to complete the cards and return them to the school.

Approximately 35,000 cards were distributed and 26,146 were returned; of these, 2362 were rejected for a number of reasons, including the following: wrong age group;

TABLE II.
Accident Rates in Pre-School Children.

Type of Accident.	Accidents per 100,000 Children Exposed at Ages (Years).											
	Under One Year.		One Year.		Two Years.		Three Years.		Four Years.		Five Years.	
	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.
As a passenger in a motor vehicle ..	99	60	171	183	599	454	879	586	645	472	356	312
Motor vehicle accident as a pedestrian ..	8	43	60	35	187	113	384	359	599	340	571	283
Burns and scalds ..	758	532	1765	1315	2198	1786	1609	1417	758	1002	702	501
Falls ..	701	644	2439	1890	5388	4148	5819	4705	5435	3732	3611	2438
Poison swallowed ..	66	60	307	375	1038	510	486	236	206	66	28	57
Accident by machinery ..	16	0	111	70	289	208	365	255	299	180	178	104
Near drowning ..	25	25	119	131	318	321	299	255	299	132	187	85
Injury by an animal ..	41	0	94	87	299	359	309	255	234	217	131	129
Foreign body in an orifice ..	82	77	119	139	215	198	281	368	206	142	150	94
Crushed by a falling object ..	57	34	77	226	374	265	337	359	187	312	197	189
Struck by a flying object ..	41	60	85	114	384	312	440	472	711	529	617	321
Cut by sharp or piercing instrument ..	91	17	128	122	533	302	917	576	1066	576	833	416

TABLE III.
Distribution of Accidents by Type.

Type of Accident.	Percentage of Each Type of Accident at Each Year of Age.											
	Under One Year.		One Year.		Two Years.		Three Years.		Four Years.		Five Years.	
	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.
As a passenger in a motor vehicle ..	5.0	3.9	3.1	3.9	5.1	5.1	7.3	5.9	6.1	6.1	4.7	6.3
Motor vehicle accident as a pedestrian ..	0.4	2.8	1.1	0.7	1.6	1.3	3.2	3.6	5.0	4.4	7.5	5.8
Burns and scalds ..	38.2	34.2	32.3	28.0	18.6	20.0	13.3	14.4	7.1	13.0	9.3	10.2
Falls ..	35.2	41.4	44.6	40.2	45.6	46.2	48.0	48.0	51.2	48.6	47.7	49.6
Poison swallowed ..	3.3	3.9	5.6	8.0	8.8	5.7	4.0	2.4	1.9	0.8	0.4	1.2
Accidents by machinery ..	0.8	0.0	2.0	1.9	2.5	2.2	3.0	2.6	2.8	2.3	2.4	2.1
Near drowning ..	1.2	1.7	2.2	2.8	2.7	3.6	2.5	2.6	2.8	1.7	2.5	1.7
Injury by an animal ..	2.1	0.0	1.7	1.8	2.5	4.0	2.5	2.6	2.2	2.8	1.7	2.5
Foreign body in an orifice ..	4.1	4.9	2.2	2.9	1.8	2.1	2.3	3.7	1.9	1.8	2.0	1.9
Crushed by a falling object ..	2.9	2.2	1.4	4.8	3.2	2.9	2.8	3.6	1.7	4.1	2.6	3.8
Struck by flying object ..	2.1	3.9	1.5	2.4	3.2	3.5	3.6	4.8	6.7	6.9	8.2	6.5
Cut by sharp or piercing instrument ..	4.6	1.1	2.3	2.6	4.4	3.4	7.5	5.8	10.0	7.5	11.0	8.4
Total percentage ..	100	100	100	100	100	100	100	100	100	100	100	100
Total number of accidents at age ..	241	181	642	538	1264	950	1296	1042	1138	815	808	521

incompleteness; omission of age. Finally 23,784 cards (12,131 for boys and 11,653 for girls) were available for analysis.

Preliminary Analysis of the Data.

Since this was retrospective inquiry, the time interval between the occurrence of an accident and the moment of recording the event on the card could influence the accuracy of the record. The age of the children at the time of recording extended from three to eight years. Thus some parents were asked to record events which had happened four years previously, and others would have to recall accidents which had occurred eight years earlier. It was decided that before all the cards for each sex could be analysed together, it was necessary to make a preliminary analysis of the cards according to the age of the child at the time when the record was made. In the first instance this was limited to the cards for boys.

Rates for each type of accident were struck and a comparison was made of these for the different ages of recording. Distinct gradients for the rates for all types of accident were found, the highest values being obtained for the younger age groups at the time of recording, with the lowest values for the boys aged seven and eight years when the record was made. This finding suggests that a

certain number of accidents had been "forgotten" by the parents of older children if the time interval was long, particularly if the accident had been trivial. This will be discussed more fully in later papers in this series in which the individual types of accident are considered.

The rates for falls at each age at which the accidents happened, and for burns and scalds to children aged one and two years, were statistically significant between those for the youngest and oldest age groups at the time of recording. The differences between the rates for other accidents and for burns and scalds at other ages were not statistically significant. Despite these differences in rates, it was decided to pool the cards. The rates will probably err on the side of understatement. Apart from materially reducing the volume of figures in the final analyses, this procedure ensured relatively large numbers in any type of accident for each year of age in which it happened.

Results.

The material presented in this paper is limited to the numbers of each type of accident at each age, and some statistical treatment of these data. Other papers in this series, which will deal with the specific type of accident, will give details about the manner in which the accident happened, the number of mild or serious accidents, and

TABLE IV.
Distribution of Types of Accident by Age.

Type of Accident.	Sex.	Total Number.	Percentages of the Same Accident by Year of Age.					
			Under One Year.	One Year.	Two Years.	Three Years.	Four Years.	Five Years.
As a passenger in a motor vehicle ..	M.	297	4.0	6.7	21.5	31.6	23.3	12.9
	F.	221	3.2	9.5	21.7	28.0	22.6	15.0
Motor vehicle accident as a pedestrian ..	M.	194	0.5	3.6	10.3	21.2	32.9	31.5
	F.	125	4.0	3.2	9.6	30.4	28.8	24.0
Burns and scalds ..	M.	862	10.7	24.0	27.3	19.9	9.4	8.7
	F.	711	8.7	21.2	26.6	21.0	14.0	7.6
Falls ..	M.	2536	3.4	11.3	22.7	24.5	22.9	15.2
	F.	1882	4.0	11.5	23.3	26.6	20.9	13.7
Poison swallowed ..	M.	232	3.4	15.5	47.8	22.4	9.5	1.4
	F.	142	4.9	30.3	38.0	17.6	4.9	4.3
Accident by machinery ..	M.	136	1.5	9.6	22.8	28.7	23.5	13.9
	F.	87	0.0	9.2	25.3	31.1	21.8	12.6
Near drowning ..	M.	135	2.2	10.4	25.2	23.7	23.7	14.8
	F.	102	2.9	14.7	33.3	26.5	13.8	8.8
Injury by an animal ..	M.	120	4.2	9.1	26.7	27.5	20.8	11.7
	F.	111	0.0	9.0	34.3	24.3	20.7	13.7
Foreign body in an orifice ..	M.	115	8.7	12.2	20.0	26.1	19.1	11.9
	F.	110	8.2	14.5	19.1	35.5	13.6	9.1
Crushed by a falling object ..	M.	133	5.3	6.8	30.0	27.1	15.0	15.8
	F.	149	2.7	17.5	18.8	25.5	22.1	13.4
Struck by flying object ..	M.	245	2.0	4.1	16.7	19.3	31.0	26.9
	F.	193	3.6	6.7	17.2	25.9	29.0	17.6
Cut by sharp or piercing instruments ..	M.	394	2.9	3.9	14.8	25.5	29.7	23.2
	F.	214	0.9	6.6	14.9	28.5	28.5	20.6

TABLE V.
Distribution of Deaths by Types for Australia, 1950 to 1952.

Type of Accident.	Percentages of Deaths Due to Each Type of Accident at Each Year of Age.									
	Under One Year.		One Year.		Two Years.		Three Years.		Four Years.	
	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.
As a passenger in a motor vehicle	25.6	22.6	7.1	11.7	6.4	14.0	19.1	16.3	11.8	15.6
As a pedestrian in a motor accident	—	—	8.4	9.7	18.4	15.8	19.1	30.3	42.7	31.3
Burns and scalds	30.2	22.6	19.4	13.6	18.4	19.3	3.5	16.3	7.4	15.6
Falls	16.3	25.8	3.2	3.9	3.7	3.5	2.4	2.3	8.8	0.0
Poisoning	4.7	3.2	19.4	15.5	3.7	19.3	4.8	9.3	2.9	6.3
Machinery	—	—	—	2.9	—	3.5	1.2	2.3	—	6.2
Cutting and piercing instruments	—	—	1.9	—	1.8	—	1.2	2.3	—	—
Injury by animal or bite or sting	—	3.2	0.6	0.9	1.8	3.5	1.2	2.3	2.9	—
Drowning	23.2	22.6	40.0	41.7	45.8	21.1	47.5	18.6	23.5	25.0

the outcome. These facts will be used to make suggestions regarding prevention, which can be more satisfactorily discussed in respect of a specific type of accident than about accidents in general.

Of the 12,131 cards returned from boys 6138 (50.5%), and of the 11,653 cards from girls 6910 (59.3%) showed that the child had not suffered an accident of a sufficiently serious nature to warrant the mother's recording it. A number of children suffered several accidents at different ages; this will be discussed in a later paper in this series.

Table I shows the numbers of each of the eight types of accident arranged according to the year of age of the children when the accident occurred. In the same table in the last line are shown the percentages of children at each age who sustained an accident in that year of age. Table II gives the rates per 100,000 children exposed for each type of accident at each year of age. The distribution of accidents, by type for each year of age, is shown in Table III, and the distributions of each type of accident over the six years (of age) investigated are shown in Table IV.

None of the accidents recorded in this study were fatal, some were mild, others were serious. Since the difference between a fatal accident and a minor accident of the same type in the same situation is often a matter of good fortune, it was decided to include here data for deaths from the same types of accidents which happened to children in the same age groups, in Australia, for the years 1950-1952, part of the period covered by the survey. Similar information in respect of earlier periods (1908 to 1950) has already been given in an earlier paper (Clements, 1952).

The percentages of deaths due to each type of accident at each year of age (0 to four years) are shown in Table V, and the distributions of deaths due to each type of accident over five years of age for which individual records are available are given in Table VI. (In the official records, deaths of children aged five years are included in the group total five to nine years of age.)

Discussion.

An interesting feature of this survey was the high percentage of children who were recorded as having had an accident. It is true that many of the accidents, particularly the falls and cuts, were relatively minor. Trivial accidents were neglected—treated as "no accident". The criterion of retention of a card in the accident group was that, so far as could be ascertained from the description, the accident was serious or could have been serious in slightly different circumstances. This criterion was not applied too rigidly since, as the survey was primarily designed to collect information about accidents with the object of developing educational campaigns, any relevant information might be of use.

A comparison of Tables I to IV with Tables V and VI shows that a number of types of accident listed in the former are not represented in the latter. These types of accident seldom result in death, and the occasional death that does occur from one of these causes is listed in the official records under "Accidents other than unspecified". Another group of accidents (accidents by machinery; accidents by cutting and piercing instruments; and animal and insect bites) are listed in both the figures for the survey and the deaths from accidents, but are relatively unimportant causes of death in children. The figures in the six tables clearly indicate that for all age groups there are five important types of accident—namely, traffic accidents, burns and scalds, falls, poisoning and near drowning or drowning. Accidents in these five categories account for between 70% and 85% of all accidents recorded, the higher percentages being in the younger age groups. This confirms the well known fact that as children grow older they engage in more diverse activities, each with their attendant accident hazards, and thus it is that the accident rate for injuries by machinery, piercing instruments *et cetera* rises. An immediate impression from Tables II, III, V and VI is that infant morbidity and mortality from accidents are low in relation to morbidity and mortality from other causes at the same age.

TABLE VI.
Distribution of Types of Deaths from Accident by Year of Age.

Type of Accident.	Sex.	Total Numbers.	Percentages of Deaths from the Same Accident by Year of Age.				
			Under One Year.	One Year.	Two Years.	Three Years.	Four Years.
As passenger in a motor vehicle	M.	53	20.7	20.7	13.2	30.3	15.1
	F.	39	17.9	30.9	20.5	17.9	12.8
As a pedestrian in a motor accident	M.	78	0.0	16.7	25.6	20.5	37.2
	F.	42	0.0	23.8	21.4	31.0	23.8
Burns and scalds	M.	71	18.3	42.3	28.2	4.2	7.0
	F.	44	15.9	31.8	25.0	15.9	11.4
Accidental falls	M.	24	29.3	20.8	16.6	8.3	25.0
	F.	15	53.3	26.7	13.3	6.7	—
Poisoning	M.	42	4.8	71.4	9.5	9.5	4.8
	F.	34	2.9	47.1	32.4	11.8	5.8
Drowning	M.	178	5.6	34.8	28.1	22.5	9.0
	F.	78	8.9	55.1	15.4	10.3	10.3

Another observation of interest is the dominance of the accident rates for males. However, this is not uniform. Where the female rates are greater than the male rates, the numbers of these types of accidents are small, and a few more accidents in either group could alter the rates between the sexes. Falls dominate the accident picture, accounting for between 35% and 51% of all accidents; however, deaths from falls do not occupy such a prominent position in the lists of accidental deaths, except in infancy. This difference is not unexpected, for common experience confirms that children have a large number of falls, and although some of these are of a serious character, resulting in a fractured limb or similar injury, they do not under normal circumstances threaten life. The figures in Tables III and V for the percentage of total accidents due to falls in infancy and for the percentage of deaths in the same age group due to falls are misleading. From reference to Tables I and II, and from data on death rates not quoted here, it is evident that accidental falls occur from three to five times more frequently in the older age groups than in infancy.

The high rates for accidents due to burns and scalds in the first three years of life, and the relatively high percentages of deaths due to these causes in the first three years of life, confirm the hazards for young children in the bathroom, kitchen and laundry. For both boys and girls one year of age, nearly one-third of all accidents are due to burns and scalds. The rates for these causes are actually higher in children aged two years; but the position of burns and scalds relative to other causes is lower because of the great increase in the rates for accidents due to falls. For children aged one year, the ratio of falls to burns and scalds is approximately 1:1.6; for children aged two years, the ratio is approximately 1:2.5. After this age both the ratio and the percentages of total accidents due to burns and scalds drop. The reasons why the second and third year of life have such a high incidence of accidents and deaths due to burns and scalds will be discussed in a later paper.

Road accidents do not occupy a prominent place in the total pattern of accidents at any of the ages under consideration. At none of these ages do the combined rates for traffic accidents to children exceed 12% of the total, either when the child is a passenger in a motor vehicle or when he is a pedestrian. Once again this is due in a large measure to the dominance of falls.

The accident rates for children who sustain accidents when passengers in a motor vehicle rise to a peak at the age of three years, with a decline in the ages of four and five years. This is unexpected; but detailed analysis of the figures shows that opening the motor-car door by children when the vehicle is in motion and thereby falling out, and also standing up without adequate support, figure more prominently as causes of accidents at this age than when the child is older.

Rates for accidents to children when they are pedestrians do not follow the same pattern for boys and girls; for boys the rates rise from just above zero in the first year of life to peaks at the ages of four and five years; for girls the peak rate is reached at the age of three years, with declines in the next two years. However, these differences in rates are not statistically significant.

Accidental poisoning occupies a relatively minor place in the total pattern of accidents. At no age for either sex does the accident rate exceed 9% of the total accidents at that age. However, deaths from accidental poisoning (Table V) do constitute nearly 19% and 16% of the deaths of boys and girls respectively aged one year, and 4% and 19% respectively of the deaths of boys and girls aged two years. Some of these differences between accident rates and death rates could be due to the unduly large number of accidents due to falls at both these ages. The rates for accidental poisoning have peaks for both girls and boys at the age of two years, although girls aged one year also have high rates. It is interesting to note that the death rates for poisoning have pronounced peaks for both boys and girls at the age of one year. Although the children covered by the survey represent a small sample

of the total population to which the death rates apply, it is probably justifiable to discuss the two sets of figures as if they were more closely related. From Table IV it can be seen that 15% of the boys and 30% of the girls who swallowed poison were aged one year at the time, and that 48% of the boys and 38% of the girls were aged two years. However, from Table VI it will be seen that 71% of the boys and 47% of the girls who died from accidental poisoning were aged one year, and that 9.5% of the boys and 32% of the girls were aged two years. The combination of these figures suggests a higher case mortality rate at the age of one year, compared with the subsequent ages. This raises the question whether young children are less able to withstand the effects of poisons than older children. The clinical experience would suggest that this is so. Another question which needs to be considered is the length of time between the swallowing of a poison and detection of the act by the mother; for a variety of physiological reasons this may be greater in younger children.

Drowning and near-drowning present an interesting set of figures. Death from drowning is the most important cause of accidental death in the first three years of life for boys, and in the first four years of life for girls; at other ages it is exceeded only by deaths from the combined road accidents. From Table III it is obvious that situations in which a child is in danger of being drowned, but is rescued in time, constitute a small percentage of all accidents at each year of age. One inference that might be drawn from these facts is that the chances of a child's drowning when it is in a situation in which it might drown are considerably greater than the chances of a child's dying from any other type of accident. Another possible explanation is that a number of children who get into a situation which in certain circumstances leads to death by drowning, extricate themselves or are rescued by their playmates without any other signs than wet clothes, and thus the mother may be unaware of the hazard in which the child has been. This is in contrast with the circumstances surrounding almost any other type of accident.

The other types of accident do not call for particular comment at this stage. However, they will be discussed in detail in a later paper in this series.

Summary.

The outline is given of a survey made of 23,784 school and pre-school children, designed to collect information about accidents sustained by these children during the first six years of their lives. The survey cards were distributed to parents through kindergartens in Australia and schools in New South Wales.

Approximately 50% of the cards from 12,131 boys and 40% of the cards from 11,653 girls recorded one or more accidents. The percentage of children who sustained an accident at each year of age rose from less than 2% in the under one year group to 12.1% for boys and 9.8% for girls aged three years, with lower figures for both sexes at the ages of four and five years.

Falls were by far the most common cause of accidents at each age; the other major causes of accidents were burns and scalds, traffic accidents, poisoning and near-drowning. These types of accidents accounted for between 70% and 85% of all accidents. Less frequent causes of accidents were accidents from machinery, injury by an animal, foreign body in an orifice, crushing by a falling object, being struck by a flying object, being cut by a sharp or piercing instrument.

Acknowledgements.

I have to thank Dr. H. O. Lancaster for statistical advice, and Mrs. Goldstein, Miss M. Reid, Miss N. Holloway and Miss M. George for valuable assistance in the sorting and analysis of the cards. My thanks are also due to the many kindergarten directors and school teachers whose enthusiasm and application made this survey possible.

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Reports of Cases.

GASTRO-ILEOSTOMY.

By THOMAS F. ROSE.

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GASTRO-ILEOSTOMY, a strange surgical error which occasionally occurred in the days of universal gastro-enterostomies, is even now found after partial gastrectomies (Moretz, 1949; Castleton and Bailey, 1950; Landry, 1951; McKenzie and Robertson, 1953).

This error may cause little harm when the duodenum is patent, as in a patient reported by McKenzie and Robertson (1953), who survived such an insult for thirty-six years with no adverse symptoms. It was in fact a surprise autopsy finding. However, if a gastro-ileostomy is made during gastro-enterostomies with a closed duodenum, or during gastrectomies, this mistake leads to most severe metabolic disturbances, causing a condition known as "ileojejunal insufficiency" (Cropper and Houghton, 1950). This is because the gastric contents are emptied straight into the terminal portion of the ileum, so that the small bowel, the bile and the secretions of the pancreas are short-circuited. In addition, a gastro-ileal or ileal ulcer may be caused (Smith and Rivers, 1943).

Clinical Record.

The patient, a female, aged sixty-six years, was admitted to the Royal North Shore Hospital because of a crack fracture of the left great trochanter sustained on the previous day. It was then seen that not only was the patient extremely emaciated, but that every meal was followed by uncontrollable diarrhoea. She herself was confused and unable to give her previous history. Inquiry from her relatives revealed that some three months previously the patient had undergone elsewhere a partial gastrectomy for a duodenal ulcer causing stenosis. The operation, whilst successful in stopping her vomiting, was followed within a few days by attacks of diarrhoea which were so sudden and unheralded that she became incontinent of faeces. These attacks would occur at any time, but were especially severe just after the eating of solid food. The abdomen became distended after meals, and this distension was relieved by the passage of the stools. At no time was there any abdominal pain. This illness had become progressively worse, so that she would not eat solid food or drink even milk because of the subsequent diarrhoea. She was able to take only water without a subsequent bowel action. Consequently the patient lost three stone in weight. There was no history of tetany or of swelling of the soft tissues or abdomen.

Examination disclosed her to be a very emaciated woman, ill, apathetic and weak (Figure 1). Mentally she was depressed and confused. Her breath was offensive, her tongue was dry but otherwise normal, and her temperature, pulse rate and respiratory rate were normal, as was her blood pressure. There was no anasarca, and no evidence of tetany, apparent or latent. The abdomen was scaphoid, with a very small, well-healed upper right paramedian incision. No abnormality was found on abdominal examination.

Observation showed that as soon as any food was taken the patient had an attack of uncontrollable diarrhoea. The faeces were fluid and yellow in colour, and at times undigested vegetables and even meat from the previous meal were passed. When fluids only were given, except milk, the diarrhoea stopped.

A barium meal was administered, and X-ray examination showed that the stomach emptied into the terminal portion of the ileum and the caecum filled within a few minutes. X-ray examination after a barium enema revealed almost immediate filling of the stomach, as the ileo-caecal valve was incompetent. The patient also vomited the barium enema as soon as it reached the stomach.

A diagnosis of gastro-ileostomy was therefore made.

Blood examination revealed results quite at variance with her clinical condition of severe starvation. A microcytic anaemia (3,500,000 red blood cells per cubic millimetre) was present, but the serum proteins were in normal concentration, and the albumin-globulin ratio was normal. The blood electrolytic concentrations were normal by photometric analysis except that for serum potassium, which was low (3.5 milliequivalents per litre). This was confirmed by electrocardiography, which showed the S-T depression of hypopotassaemia. Radiographic examination of the pelvis and of the upper parts of the femora revealed a crack fracture of the left great trochanter. There was no evidence of any bone decalcification more than usual for the patient's age.

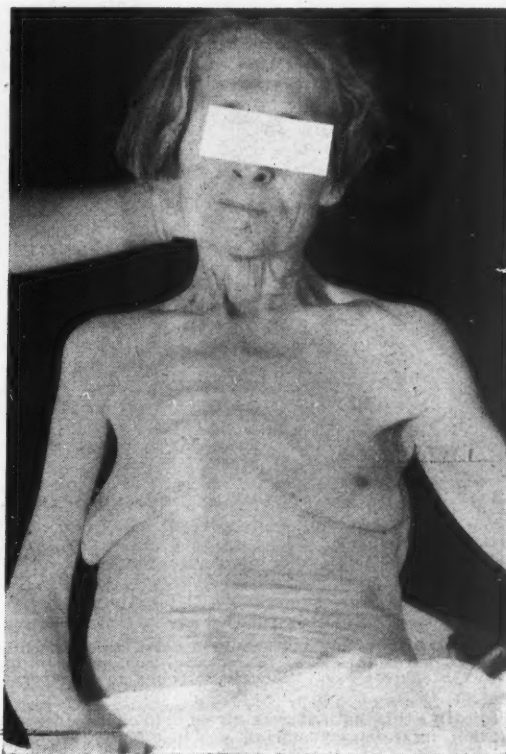


FIGURE 1.

Photograph of the patient showing the emaciation and the small incision through which a gastrectomy was performed.

After preparation with blood transfusions and parenteral fluid and potassium therapy the patient's condition improved so that the abdomen was able to be explored. A partial gastrectomy had been performed, the end of the stomach having been anastomosed in front of the transverse colon to the side of the ileum only six inches from the caecum (Figure 2). There was no ulceration of the ileum or the stoma. The anastomosis was undone, the hole in the ileum was sutured, and a correct antecolic anastomosis was performed between the stomach remnant and a proximal jejunal loop. However, the patient went into a state of apnoea the following day and after some hours died. An autopsy was not permitted.

Discussion.

Patients with gastro-ileal anastomosis after a partial gastrectomy present a clinical picture not unlike that of a gastro-jejunoileal fistula caused by an anastomotic or

jejunal ulcer after a posterior gastro-enterostomy. However, the symptoms of the former occur almost immediately after operation (as soon as solid food is taken) and no abdominal pain is present. The latter occurs after a certain post-operative interval, and is characterized first by the appearance of pain due to the jejunal or stomal ulcer, and then by the appearance of symptoms due to the fistula, often with disappearance of the pain.

An awareness of this condition is the first step in the diagnosis, for the frequency of defecation and the character of the stools containing undigested food are quite unlike those found after some normal gastrectomies, when frequent loose motions may be present. Owing to the virtual elimination of the secretions of the small bowel, pancreas and liver, there is often an excessive amount of fat in the stools. (Unfortunately, this aspect was not investigated in the case under discussion.) This ileo-jejunal insufficiency leads to a metabolic syndrome not unlike sprue (Cropper and Houghton, 1950) or massive resection of the small bowel (Cosh, 1944; Holman, 1944).

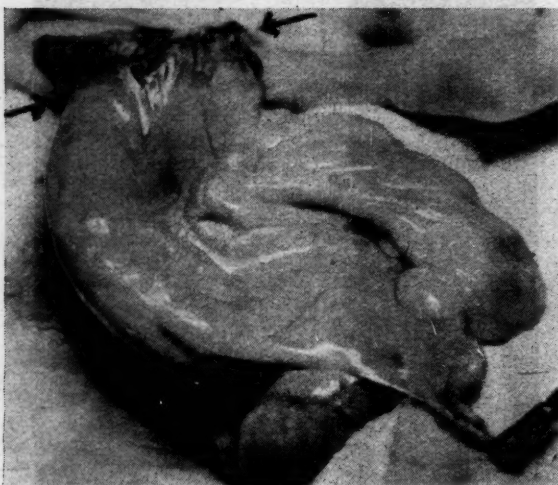


FIGURE II.

The arrows point to the anastomosis between the stomach and ileum, the left-hand one to the efferent loop, the right-hand one to the afferent loop. The sucker points to the appendix.

Clinically, this patient was emaciated owing to starvation; but interestingly enough she had no lowered blood protein levels, except as shown by her anemia, and this was the reason why she had no anasarca. Similarly, her electrolyte concentrations were normal except for the low potassium serum level, which probably played a great part in her apathy and weakness. However, her condition was such that, although parenteral therapy, including blood transfusions, enabled her to undergo a laparotomy with the performance of a comparatively small operation, she died the following day.

It is of interest to speculate how this surgical accident could occur. In looking at Figure I it may be seen that the gastrectomy had been performed through a tiny incision, and I think that this may have prevented the surgeon from adequately exploring the small bowel to select the correct jejunal loop. Only in this way, surely, could the duodeno-jejunal flexure have been missed, as it is always comparatively easy to find. This case thus points a moral to all would-be gastrectomists and shows the dangers of small incisions.

Summary.

The clinical record is presented of a patient who underwent a partial gastrectomy, in which the stomach was anastomosed to the terminal portion of the ileum.

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A CASE OF NEUROBLASTOMA.

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In a recent review of neuroblastoma, Ralph Phillips (1953) offers a more hopeful prognosis with modern therapy than in the syndromes described by Pepper and Hutchison. The syndromes described by these two authors were considered to be universally fatal, and this idea has become firmly entrenched. Phillips considers that if the natural exuberant growth of these anaplastic tumours can be restricted by therapy, a spontaneous differentiation of the original tumour occurs, and regression of the visceral and skeletal metastases is often seen. Willis (1948) also states that many of these tumours show all combinations of and transitions between fully anaplastic neuroblastoma and fully mature ganglioneuroma, and that the neuroblastoma occurs in infants and the more mature tumours occur in the older age groups.

On the other hand, a more recent paper from America by Oberkircher, Straubitz and Parmenter (1953) comes to an opposite conclusion; these authors consider that although in an occasional case recovery may occur, most are rapidly fatal. The therapy used by them was nitrogen mustard and irradiation, together with surgical removal of the primary mass. They did not consider that this therapy affected the outcome.

The following case is reported as typical of these anaplastic tumours.

Clinical History.

A, aged two years, was first examined in December, 1953. He was the youngest of three boys, his two brothers, aged four years and six years, being quite well and having suffered from no previous illness. Both his parents were normal and did not give any history of previous illness. The patient had suffered from recurrent attacks of bronchopneumonia since birth. Three months before examination, glands were noticed on the left side of the neck, which were slowly increasing in size. An attack of tonsillitis followed, and the glands increased in size rapidly after this. The throat condition settled down with antibiotics, but the glands continued to increase in size. The glands on the opposite side of the neck were felt about six weeks later.

On examination, the patient was a well developed infant. His tonsils were enlarged and cryptic and there were multiple regular, firm, mobile, discrete glands in the left side of the neck, from 3.5 centimetres in diameter, extending into the upper anterior and posterior triangles and down to the clavicle. A few small glands were present in the tonsil area and the upper posterior triangle on the right side to 0.75 centimetre in diameter. No other glands were detected. There were no other abnormal findings at the physical examination.

The blood count gave the following information: haemoglobin value was 92%, and the white blood cells numbered 3600 per cubic millimetre, 57% being neutrophile cells, 37%

lymphocytes, 1% monocytes, 3% eosinophile cells and 2% basophile cells. A small gland was removed from the neck for biopsy and the pathologist reported as follows:

Section of the lymph nodes shows several clumps of anaplastic sphenoidal cell carcinoma growing from the periferal sinuses. In parts the tumour has necrosed. The carcinoma cells are large with scanty cytoplasm and show many mitoses.

From February 16 to February 22, 1954, X-ray therapy was given to the neck, the total dosage being 672r. The glands disappeared after therapy.

On March 31, when he was examined again, the child seemed weak when walking. He had bruises around both orbits and rounded tumours on the skull. X-ray examination of the bones at this time showed rarefactions in the diploë of the skull with periosteal elevation under the scalp tumours. Gross bony changes were present in the pelvis, particularly in the ischium and in the right femur, where periostitis was also present. A further course of X-ray therapy was commenced to the left side of the neck for growth restraint purposes. Four doses of 150r were given between April 4 and April 13.

A blood examination on April 6, 1954, gave the following results: the hæmoglobin value was 8.5 grammes per centum (55%) and the red blood cells numbered 2,740,000 per cubic millimetre, the white blood cells 4250 per cubic millimetre and the platelets 100,000 per cubic millimetre. Of the white blood cells, 50% were polymorphonuclear cells, 42% lymphocytes, 6% monocytes and 2% eosinophile cells. The report on the blood film was as follows:

In view of the steep fall in the hæmoglobin, the low platelets and the presence of nucleated red cells in the periferal blood it is probable that the marrow is infiltrated by the neoplastic process. Marrow puncture should be done.

The child was too ill at this time to allow the marrow puncture to be done.

His condition deteriorated, and he was admitted to the Austin Hospital on April 14. An X-ray report on April 30 was as follows:

There were diffuse changes in the ilium, ischium and pubis and both femoral shafts and necks with subperiosteal rarefaction and generalized exaggeration of the trabecula pattern without actual localized deposits. Presumably this is a diffuse secondary invasion, but radiologically it is not distinguishable from thyroid, renal or hemopoietic reaction.

No blood examination was carried out, but the child's condition deteriorated and he died on May 10.

Post-Mortem Examination.

The body was that of a well-nourished male infant of rather pale complexion, with large bilateral periorbital hæmatomata and several large, firm, nodular tumours beneath the scalp over both parietal bones.

In the left side of the neck from the basiocciput down to the axilla was a matted chain of large, firm lymph glands. The lymph nodes were quite discrete around the basiocciput, but became fixed into a hard nodular sheath completely surrounding the common carotid, and extending down to the dome of the pleura, where there was a large firm circumscribed tumour three centimetres in diameter. Extending from this into the axilla were numerous small firm lymph glands about 0.75 centimetre in diameter fixed together.

In the chest a small atrophic thymus was seen in the mediastinum, but both lungs were normal. There were large nodular masses two centimetres in diameter, one on each side of the dorsal part of the spine, that on the left covering the lateral surfaces of the fourth and fifth dorsal vertebræ, that on the right covering the lateral surfaces of the fifth and sixth dorsal vertebræ. These masses were extrapleural and firmly fixed to the bone, requiring sharp dissection to separate; but the bone cortex itself did not show any reaction. These masses appeared to be infiltrating into the neural canal through the vertebral foramina, but further dissection in this area was not carried out.

In the abdomen there were two large, rounded, discrete lymph nodes five centimetres in diameter in the right iliac fossa over the external iliac artery, and numerous small discrete lymph nodes scattered through the mesentery and the transverse mesocolon. Both suprarenals were normal in appearance, but in the upper pole of the right kidney there were numerous raised plaques which extended fairly widely into the kidney substance.

Examination of the skull showed a number of large, round, discrete nodules approximately three centimetres in diameter over both parietal bones and to some extent over the squamous portion of both temporal bones. On the right side the mass of tissue extended into the right temporal fossa and through the sphenopalatine fissure, to form a small plaque of tissue beneath the dura in the mid-cranial fossa, on the greater wing of the sphenoid. The tumour nodules over the parietal bones on both sides were firmly fixed to the bone and could be removed by sharp dissection only, a smooth surface being left; but on the internal surface the bone was raised into large bony spicules, which extended into a flat irregular plaque of tissue growing over the external surface of the dura and apparently continuous through the bone with the tumour nodules beneath the scalp.

The brain itself appeared normal.

A section of the bone was removed from the vertebral column, and a diffuse white mottling was seen in the red marrow of many of the dorsal vertebræ, although in the lumbar vertebræ the red marrow appeared normal. The upper portion of the shaft of the right femur was exposed, and the peritoneum was elevated, purulent fluid being disclosed. A small segment of the shaft was removed for examination.

Microscopic Examination of the Tissue Removed.

Microscopic examination of the paravertebral tumours revealed large irregular polygonal cells with a little cytoplasm and a large nucleus, arranged in irregular clusters with rather acellular fibrous trabeculae and reticular supporting tissue. The tumour cells were often arranged on the reticular tissue framework to give a pseudo-papillary appearance. Mitotic figures were frequent, and no recognizable tumour pattern could be made out.

The masses in the upper pole of the right kidney were small abscesses; a few nodules of tumour tissue were also present. In sections of the spleen, liver and suprarenals were seen numerous small tumour emboli in many of the capillaries, but no massive tumour growth in the parenchyma itself.

Examination of the large tumour nodules beneath the scalp revealed similar tumour structure to the paravertebral tumours, and the tumour tissue was seen to extend through the bony cortex of the skull and was widely scattered in the cancellous bone of the diploë, completely replacing the red marrow in this area.

The lymph nodes from which sections were prepared had been entirely replaced by tumour tissue. Tumour infiltration was seen also in sections of the bone marrow in the dorsal vertebræ and right femoral shaft.

Examination of sections of the thymus revealed general atrophy and no evidence of tumour growth.

The diagnosis of neuroblastoma of the paradorsal sympathetic ganglia (bilateral) was made.

Discussion.

In a Hunterian lecture on July 10, 1952, Ralph Phillips states that cancer now ranks as one of the major hazards of life for children aged under fifteen years, taking third place to accidents and pneumonia. He considers, therefore, that a more alert suspicion of cancer as a cause of obscure symptoms in children is required. Neuroblastoma, next to leuchæmia, is the commonest type of cancer in children. Oberkircher, Straubitz and Parmenter emphasize the insidious nature of the symptoms. It is agreed that diagnosis is difficult in most cases, and of the presenting symptoms, fever and anorexia were most common. Rheumatic fever, tuberculosis and Wilms's tumour were amongst the initial diagnoses. Any palpable mass must be considered

a malignant tumour until proved otherwise. Diagnosis is aided by aspiration biopsy, bone marrow aspiration and relevant X-ray examination. Excretion pyelograms are of distinct value in adrenal tumours.

Willis is particularly taken with the idea of maturation of these growths. The undifferentiated tumours are seen mainly in infants and young children, and the more mature ganglioneuromata mainly in young adults. All gradients between these two stages are often seen, with a well defined admixture of neuroblastoma with quite distinct adult nerve cells. Willis states that adult nervous tissue is incapable of growth and multiplication, so that the only explanation for the presence of ganglioneuromata is that they have originated from immature nervous tissue. Ralph Phillips has followed up this idea in a plea for intensive therapy of these anaplastic growths in children to restrict the exuberant growth until a natural maturation process can develop. He treats them by surgical removal as far as possible of the primary growth, and intensive irradiation therapy (2000 to 3000r in ten to twenty days, but larger doses may be necessary); chemotherapy also must be intense, and nitrogen mustard, anti-folic acid and cortisone are all used. His paper holds out some hope for these unfortunate patients.

Summary.

A case of neuroblastoma which terminated fatally is reported. Attention is drawn to an attitude of greater hope in the treatment of these patients.

Acknowledgement.

I wish to thank Dr. R. Kaye Scott for permission to publish this case, and Dr. R. Motteram for the biopsy report on the gland and for the blood examinations.

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SPONTANEOUS RUPTURE OF THE SPLEEN COMPLICATING INFECTIOUS MONONUCLEOSIS.

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INFECTIOUS MONONUCLEOSIS may be regarded as the "Cinderella of hæmatology"; it is only since 1920 that the hæmatological features of this disease have been recognized and studied in detail. Yet it was known in the latter part of the nineteenth century, when epidemics were reported by Pfeiffer (1889), by Park West (1896) and by Dawson Williams (1897), and it was known to be characterized by glandular enlargement and splenic enlargement. In spite of this, and of an article by J. E. Burns in 1908, which appears to have been completely ignored or overlooked, it was not until after World War I that the disease was investigated with any thoroughness. However, both the hæmatological and pathological features of this disease are now well recognized, and because the latter—and consequently the symptomatology—can be so protean, it is understandable that the diagnosis of the disease may be very difficult, particularly in isolated cases.

The symptoms of the disease, together with the wide range of complications which can occur, are all in the main due to hyperplasia of lymphoid tissue, together with the focal mononuclear infiltrations of the reticulo-endothelial system, and of the heart, liver and brain. In fact, any tissue or organ in the body may be involved (Custer and Smith, 1948). Such complications include meningo-encephalitis, myocarditis, hepatitis, peripheral neuritis, hæmoptysis, naso-pharyngeal hæmorrhages, hæmaturia, rectal hæmorrhage and rupture of the spleen.

In recent years there has been a steady increase in the number of deaths reported in infectious mononucleosis, and the most common single cause has been hæmorrhage from splenic rupture. The number of deaths from this cause reported up to 1953 was nine (Leibowitz, 1953).

The total number of cases of ruptured spleen complicating this disease is 20, as far as I can ascertain. The percentage mortality from this complication is therefore very high.

Because of the multiplicity of symptoms with the consequent difficulty in diagnosis, it was fortunate that in 1932 Paul and Bunnell observed that heterophile antibodies developed in high titre in human serum in infectious mononucleosis. With one or two minor exceptions this occurred in no other disease. These exceptions can be ruled out by the further observation that the agglutinins developed are not absorbed by guinea-pig kidney, but are absorbed by washed boiled beef red cells. This fact is important, because low titres can occur in other diseases; but the foregoing absorption pattern does not.

Sir Henry Tidy (1948) agrees with Paul and others that a positive serological reaction (Paul-Bunnell reaction) is proof of infectious mononucleosis, and a negative response does not exclude it. This serological test is of importance in diagnosing the disease, for the atypical lymphocyte characteristically seen in infectious mononucleosis is also seen in a variety of other diseases. This point has been repeatedly stressed by many authors. Virus infections, such as infectious hepatitis, virus pneumonia, rubeola, rubella, herpes zoster, herpes simplex, influenza and other respiratory infections, have all caused this type of cell to appear in the peripheral blood, as have other non-virus diseases.

As Leibowitz (1953) pointed out in his monograph on this disease, the previously time-honoured diagnostic triad of clinical, hæmatological and serological findings must now have added to it a fourth requirement—namely, evidence of liver dysfunction. This is interesting from the point of view of the jaundice which developed in the following case some three months after the onset of the illness. This was thought to be due to homologous serum jaundice; but it could be that it was a flare-up in an organ previously affected by infectious mononucleosis. Something more will be said of this later.

Clinical Record.

The patient, a single girl, aged twenty-two years, requested medical advice on June 10, 1954. She was complaining of left earache, a sore throat and a painful swollen gland on the left side of her neck. She also said that for the previous two weeks she had been feeling very run down and extremely tired, often forgoing meals so that she could lie down and rest. During this period she had been working as a school teacher.

On examination of the patient, her temperature was 97.2° F. and her blood pressure was 130 millimetres of mercury, systolic, and 80 millimetres, diastolic; her throat was slightly reddened generally, and a cervical gland on the left side was enlarged, firm and tender. The canal of her left ear was completely occluded by wax. These were the only positive findings. Examination of her cardiovascular, respiratory, gastro-intestinal and central nervous systems revealed no other abnormalities. No other abnormal glands were detected, nor was the liver or spleen palpable. The hæmoglobin value was 100%. An X-ray examination of her chest revealed no abnormality. The ear was syringed and the plug of wax removed, a slightly injected drum being revealed.

The following day the solitary cervical gland was further enlarged and more tender, and there was some increase in redness of the throat, the left faucial region having a swollen, shining appearance. It was thought that the patient might have been developing quinsy, and a course of sulphadiazine was commenced. Her temperature that evening was 99.2° F.

The next day the patient's condition was largely unchanged.

On the following day a morbilliform type of rash developed across the back of her neck and upper part of her trunk. At this stage it was thought that she could be suffering from infectious mononucleosis. Consequently a blood smear was stained with Leishmann's stain, and on examination of this slide it was seen that there were many abnormal cells of the monocytic variety (40%) and a provisional diagnosis of infectious mononucleosis was made. The sulphadiazine therapy was discontinued.

The following day the patient felt more comfortable, but complained of some tenderness when palpated over the region of the liver, which was not palpable; the spleen was not palpable, nor was there any tenderness over it. The morbilliform rash had almost completely faded.

On the sixth day after her initial visit to the surgery the writer called at the patient's home at 10 a.m. on a routine visit and found her obviously shocked and in pain. She said that one hour before she had suffered a sudden severe pain in the upper left quadrant of her abdomen. She had awakened that morning feeling better than she had done for some time and had eaten a good breakfast. She had just settled down after breakfast when the pain occurred.

On examination of the patient, her pulse rate was 112 per minute, and her blood pressure 80 millimetres of mercury, systolic, and 60 millimetres, diastolic. Tenderness was present over the left upper quadrant of her abdomen, with marked release tenderness in this region. There was no rigidity, nor had there been any shoulder pain.

A diagnosis of a ruptured spleen was made, and the patient was moved by ambulance to hospital, where on her arrival her blood pressure was recorded at 60 millimetres of mercury, systolic. A pint of serum was given immediately, intravenously, while her blood group was being determined, and this was followed by two pints of blood (A(II), Rh-negative). Shortly after the commencement of administration of the second pint of blood she had recovered sufficiently to be moved to the operating theatre.

The anaesthetic consisted of "Pentothal" induction followed by "Flaxedil" and endotracheal intubation, with maintenance of anaesthesia by means of nitrous oxide and oxygen. An upper left transverse incision was made, and two or three pints of blood were found in the peritoneal cavity. On palpation of the spleen it was about three times its normal size and had undergone a series of ruptures in the hilar region. It was delivered with difficulty because of its extreme friability, and after the gastro-splenic ligament had been secured and divided, the spleen more or less fell off its pedicle; this was secured with some difficulty and haemostasis was effected. The presence of two or three spleniculi in the neighbourhood of the spleen was noted, and these had the same swollen appearance as the spleen. They were left *in situ*. The abdomen was closed without drainage. During the operation the patient received a further one and a half pints of blood, making a total of three pints of blood and one pint of serum. Her general condition on her return to the ward was good.

She received a further two pints of blood in the first twenty-four hours after operation, and she was maintained by intravenous fluid therapy with gastric aspiration for three days. The haemoglobin level on the day after operation was 80%, and the pulse and blood pressure were steady and satisfactory.

Her immediate post-operative convalescence was uneventful, with the aid of penicillin and streptomycin, and she was discharged home after two and a half weeks in hospital.

The writer's original slides, together with fresh ones and a specimen of blood taken on the second post-operative day, were dispatched to the pathology department of the Alfred Hospital, Melbourne, for a Paul-Bunnell test and confirmation of the diagnosis.

The patient's convalescence continued satisfactorily for three weeks, but was then interrupted by periods of malaise and lethargy for which no cause could be found.

The result of the Paul-Bunnell test reverted to negative within the month, and the blood picture also gradually returned to normal. In view of this, the clinical condition of the patient was thought to be due to so-called post-splenectomy syndrome rather than to a continuance of the glandular fever. Treatment over this period was symptomatic, apart from iron given by mouth to encourage the return of her haemoglobin to 100%.

This state of affairs continued for almost three months, with but very gradual improvement. The patient would feel well for three or four days, and then would undergo a period of two or three days of extreme fatigue with some odd aches and pains and general malaise. At no stage did she show a temperature rise.

Exactly three months after operation (September 17, 1954) the patient was completely prostrated again, far more than at any time over the preceding two months. On examination of the patient, the only positive findings were dark urine and the fact that her faeces that morning had been clay-coloured. The urine gave positive results to Fouchet's test and Hay's test for bile pigments and bile salts. She had had no pain of any type, nor was there any tenderness over the liver, which was not enlarged. Examination of a blood film at this time revealed an almost normal picture.

In view of the time factor since blood transfusion, together with the negative evidence concerning both glandular fever and infective hepatitis, it was thought that the patient was developing homologous serum jaundice following transfusions three months beforehand. Jaundice showed clinically the next day and persisted for three or four days before rapidly fading. Her appetite returned on the third day and she was given a diet of low fat content supplemented by "Meonine" and vitamin B group tablets. Over the following three weeks she maintained a slow but steady improvement, and at the present time this improvement continues, but she is not yet back to full activity. The latest liver function tests, five months after operation and two months after the jaundice, still show a "+" response to the cephalin flocculation test.

Results of Investigations.

A number of investigations were carried out, with the following results.

On June 13 examination of a blood film showed that the erythrocytes and platelets were normal. The appearances were consistent with leucocytosis due to an increase in the lymphocyte-monocyte series, many of which were atypical in appearance and were identical with those seen in infectious mononucleosis. A differential leucocyte count gave the following results: monocyte series 40%, polymorphonuclear cells 30%, lymphocytes 28%, eosinophilic cells 2%.

The Paul-Bunnell test was carried out on three occasions. On June 18 the result was positive (titre 1 in 56). Agglutinins were not absorbed by guinea-pig extract (titre 1 in 28) but were absorbed by ox red blood cells extract (titre 1 in 14). On July 14 the result was negative (titre 1 in 14); agglutinins were absorbed by ox red blood cell extract and by guinea-pig kidney extract. On August 25 the result was negative (titre 1 in 7). Agglutinins were absorbed by guinea-pig kidney extract and by ox red blood cell extract.

On June 16 a section of spleen was examined. The normal architecture of white and red pulp was preserved; however, the red pulp showed an increase in nucleated cells, many of which appeared similar to the abnormal white cells in the peripheral blood. These cells were present in the walls of the sinusoids as well as in the lumen. Otherwise the spleen appeared to be normal.

Liver function tests were carried out on two occasions. On September 21 the total serum protein content was 6.7 grammes per centum (albumin 3.6, globulin 3.1, grammes per centum). The serum alkaline phosphatase was 29 King-Armstrong units, and the serum bilirubin content was 4.5 milligrammes per 100 cubic millimetres. On November 17 the total serum protein content was 6.3 grammes per

centum (albumin 4.2, globulin 2.1, grammes per centum). The serum alkaline phosphatase content was 11 King-Armstrong units, the serum bilirubin content was 0.25 milligramme per 100 cubic millimetres, and the cephalin flocculation test produced a positive ("4") result.

The haemoglobin value was 80% on June 18, 95% on September 29, and 97% on November 16.

Discussion.

Belton points out the rarity of this complication, together with the fact that the diagnosis of the splenic rupture is usually made late. However, in the present case severity of the symptoms at the onset of the rupture and the unmistakable clinical signs led to an early diagnosis.

The other feature of the case was the fact that, as far as can be ascertained, the rupture of the spleen was truly spontaneous. It is well recognized that slight trauma, such as excessive palpation of the area, can be responsible for rupture of these enlarged, extremely friable spleens; bending forward of the trunk or straining at stool may also be sufficient. From what the writer can ascertain, this patient was lying quietly in bed at the time; therefore, the rupture was apparently spontaneous. In view of the condition of this spleen revealed at operation, it would appear that the mere act of breathing might be all that was needed to precipitate the rupture of the capsule, the splenic tissue was so friable and pulpy. Hence the term "spontaneous" is merely a relative one.

It may be said that both the patient and her medical attendant were extremely fortunate in that two pints of A(II) Rh-negative blood had arrived by air from Melbourne that morning, having been sent for an elective transfusion to another patient. This, with blood from a local donor, was sufficient to keep the patient going until the Red Cross Society could send further supplies to Swan Hill.

Acknowledgements.

The writer wishes to thank Dr. R. Hayes and Dr. A. W. Jackson, of the Alfred Hospital, for their cooperation in having the various tests done, particularly as the distance involved was an added handicap. Thanks are also due to Dr. H. H. Martin and Dr. H. G. Stevenson, of Swan Hill, for their willing help and advice, both with the operative treatment and general management of the patient and with the writing of this article.

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LYMPHADENOSIS AMONGST NEW GUINEA NATIVES.

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LITTLE has been reported on the pathology of blood disorders in primitive people, and especially in the Melanesian race. It appears that the following cases are worth recording.

Case 1.

A male native of Rabaul Sub-District, aged approximately twenty-two years, was admitted to the hospital on October 25, 1951. He had noticed swelling in the neck and under both armpits approximately three years prior to his admission to hospital; however, it had caused no dis-

comfort. Recently he had developed a large "lump" in the abdomen, had had a high temperature and had lost weight. On his admission to hospital he complained of vomiting and watery motions.

On examination of the patient the mucous membranes of the mouth were conspicuously pale. There were ulcerating patches of buccal mucosa of irregular outline. Both tonsils were enlarged. Tubular breathing was present over both lung fields on auscultation, and increased dullness over the mediastinum on percussion. The abdomen was slightly distended and tender on palpation. The spleen occupied the entire left subcostal region and left iliac fossa. It had a smooth surface and well-rounded edges. The liver was felt four fingers' breadth below the right costal margin. The retroauricular, cervical, supraclavicular, infraclavicular, axillary, cubital and inguinal lymph nodes were uniformly enlarged and firmly attached to the surrounding tissue. The nodes were of firm elastic consistency. The covering skin was of normal appearance. There were no other detectable infiltrations of the skin. A blood examination on October 27 gave the following information: the haemoglobin value was 41% and the erythrocytes numbered 2,000,000 per cubic millimetre; the leucocytes numbered 90,000 per cubic millimetre, 2% being neutrophil cells and 98% lymphocytes; there were no basophile or eosinophile cells and no monocytes. The erythrocyte sedimentation rate was 125 millimetres in the first hour (Westergren). The coagulation time was fifteen and a half minutes. On October 26 a skiagram of the chest showed enlarged perihilar shadows, distinct bronchovascular markings and a funnel-shaped area of density in the mediastinum. The diagnosis of chronic lymphatic leukaemia was made. The patient was treated with Fowler's solution (*Liquor Arsenicalis*), procaine penicillin and sedatives. The clinical course was one of steady deterioration. A further skiagram of the chest on October 31 showed an effusion of the base of the left lung. A differential leucocyte count on November 9 presented the following entirely different picture: 4.8% were segmented neutrophil cells, 36.2% band forms, 8% eosinophile cells, 2% monocytes, 23% large lymphocytes and 26% small lymphocytes; there were no basophile cells. It appeared to be a transient aleukemic phase before death. The elevated temperature did not subside. The patient had difficulty in swallowing and complained of severe itching of the skin. He could not walk because of intense pain in both legs. He died on November 14.

Autopsy Findings.

Examination of the brain and skull revealed no abnormalities. The lymph nodes in the mouth region were enlarged and confluent. There were massive pleural adhesions, especially of the left lung, and an effusion at the base of the left lung. On examination of sections no gross pathological lesions were seen.

The mediastinal, tracheo-bronchial and retrosternal lymph nodes were enlarged and of soft consistency, with jelly-like central disintegrated areas. A hemorrhage had occurred into the peritoneal cavity. The spleen was uniformly enlarged and soft. The capsule was smooth. On examination of sections the pulp was greyish-red and the architecture seemed partly obscured. The liver was enlarged and pale. The overgrowth of Peyer's patches in the intestines was pronounced. Examination of sections showed them to be of striking gelatinous appearance. The mesenteric lymph nodes were discretely enlarged. Histological examination of the lymph nodes revealed loss of normal gland architecture, which was replaced by a diffuse arrangement of round cells supported by delicate fibrillar stroma. The cells were fairly uniform in size and preponderantly mononuclear. Detailed study of nuclear pattern was not entirely satisfactory in this material, but the majority of cells resembled mature lymphocytes. Examination of sections of the biopsy specimen from a lung showed cells resembling mature lymphocytes in lymph spaces and in the adjoining stroma. Some alveoli were collapsed, but many were distended with watery exudate.

The histological picture was consistent with chronic lymphocytic leukaemia.

Case II.

A female native of Manus Island, aged approximately fifty-five years, was admitted to the hospital on June 16, 1952. She had a history of abdominal pain, occasional vomiting and diarrhoea. She had been confined to bed during the last two years. On her admission to hospital she complained of pain in the right side of the chest and the right loin.

On examination of the patient, she was seen to be in an advanced, cachectic state. The mucous membranes of the mouth were pale and cyanotic. The breath sounds were increased and there was increased dullness on percussion over the bases of both lungs. A friction rub was distinctly heard over the base of the right lung. The abdomen was tense and the upper abdominal reflexes were absent. Tenderness was elicited on palpation and percussion in the right loin. The engorged veins on the chest and abdomen produced a discrete pattern of *caput Medusae*. On palpation of the abdomen an irregularly shaped mass below the right costal margin was revealed. It occupied the right iliac fossa, reaching the iliac crest laterally, and being 138 millimetres (five and a half inches) below the xiphoid process of the sternum in the middle line. The inguinal lymph nodes were considerably enlarged. The right groin contained a mass the size of a large orange firmly attached to its base. The central area was disintegrated and formed a crater-like ulcer, discharging an odorous yellow fluid. The mass was of hard consistency. A blood examination gave the following information: the haemoglobin value was 42%, and the erythrocytes numbered 2,000,000 per cubic millimetre; the leucocytes numbered 19,000 per cubic millimetre, 32% being segmented neutrophil cells, 10% band forms, 13% eosinophil cells, 5% monocytes, and 40% lymphocytes; there were no basophil cells. The clinical course was very stormy and the patient died on June 22.

Autopsy Findings.

Autopsy revealed pleural adhesions of the right phrenicostal sinus and consolidation of the middle and lower lobes of the right lung.

Examination of sections revealed multiple, rounded, discrete nodules of solid consistency scattered throughout the lung parenchyma. The spleen was small and hard. The liver was uniformly enlarged. The cranial surface was smooth. The capsule stripped easily. The caudal surface was intimately connected with a huge mass of the pararenal space extending from the right phrenicostal sinus to the pelvis. The tumour was removed *in toto* and the separate organs were isolated. The right lobe of the liver was occupied by a solid, solitary cauliflower growth covered with a layer of liver parenchyma thirteen millimetres (half an inch) thick (see Figure I). The growth seemed to consist of large and small round confluent nodules. In some of them there was central necrosis. The periportal area was greatly invaded. The right kidney was shapelessly deformed by the invading tumour. The left kidney was small and scirrhous. The mesenteric glands were enlarged and of firm consistency. The cut surface was flesh-coloured, and in it there were green foci of necrosis.

Histological examination of the mesenteric and inguinal lymph glands showed, in each case, complete loss of normal architecture and replacement of gland structure by a neoplasm consisting of undifferentiated round cells supported by a fine reticulum. In both the kidney and the liver there were neoplastic deposits of similar pattern. In addition, there was evidence of nephrosclerosis.

The findings were consistent with a lymphosarcoma, multifocal or metastatic in lymph nodes with other metastases in the liver, kidney and lung.

Comment.

In accordance with Mulligan's (1951) classification, Case I would be considered one of neoplastic involvement of lymphoid tissue with blood-stream invasion known as

lymphocytic lymphosarcoma, and Case II one of lymphoblastic lymphosarcoma.

Pleural effusion (Case I) was an unusual complication (Twining *et alii*, 1951).

Neurological lesions were not seen. However, they are not rare (Schwab *et alii*, 1935), and according to Heinrich (1946) they occur in 18% of all cases. The haemogram in Case II was not instructive, although there was anaemia with moderate leucocytosis and neutropenia. It seems likely that the initial lesion was in the abdomen and that it resulted in widespread local metastasis and invasion of the neighbouring organs. Tischendorf (1946) points out that secondary deposits in lymphosarcoma can occur by lymphogenous as well as by haematogenous spread.

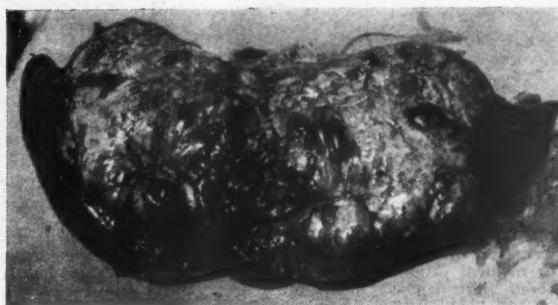


FIGURE I.
Metastatic growth of the liver (Case II).

According to De Langen and Lichtenstein (1936), lymphatic leukaemia is extremely rare in the tropics. But even in large centres, such as the London Hospital, there were only 25 cases of the disease in a period of fifteen years (1912 to 1927—Panton *et alii*, 1929).

In the Territory of Papua two cases were reported by Giblin (1920, 1921) in a period of fifteen years.

There were 31,077 admissions to Rabaul Native Hospital in the period from January 1, 1949, to May 1, 1953. Case I in the present paper was the only one of this disease (0.0032% in relation to all admissions). Unless routine hematological investigations are made on all patients with enlarged spleen, generalized lymphadenopathy and lesions in the mouth, this disease can be easily overlooked.

The most common mistaken diagnoses are those of chronic malaria, tuberculous or non-specific adenitis and deficiency diseases.

Summary.

One case of lymphocytic lymphosarcoma (chronic lymphatic leukaemia) and one case of lymphoblastic lymphosarcoma in New Guinea natives are presented with their clinical, pathological and histological findings.

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Reviews.

The Sociology of the Patient. By Earl Lomon Koos, Ph.D.: Second Edition; 1954. New York: McGraw-Hill Book Company, Incorporated. 9½" x 6½", pp. 284, with 46 illustrations. Price: \$4.50.

DR. E. L. KOOS is to be congratulated on the second edition of his book, "The Sociology of the Patient". The somewhat dry title does not do justice to the subject. "The Story of the World Around You" might have been more apt. Although written primarily for nurses, much of the book is relevant to medical students and practitioners. The author has a belief that a good nurse must understand the social life of the people with whom she is in contact. He outlines this theme in a well-written volume with copious illustrations.

It would have been better for us if the author had made the setting in Australia instead of the United States of America, but notwithstanding this, much is applicable. Perhaps he might be persuaded to visit this country and with equal clarity describe our own ecology. The expansion of population and the influx of New Australians demand a knowledge of the many aspects under discussion.

Dr. Koos commences with man as a social animal and traces his evolution through time. There is involved the meanings of culture, the changing patterns together with the significance of folkways, mores and laws. In the words of the author: "Culture is a major determining factor in man's life, for it is the accumulated ways of believing and living which form the pattern by which he lives."

There follows the story of man as a person who changes from the squalling infant into an adult human being. It involves ways of learning attitudes, social roles, motivations, individual needs in order to produce a stable personality.

The importance of family life is rightly stressed. The causes for the changing pattern in America are analysed. Industrialization, urbanization, increased mobility, changing motivations, the shift of responsibility and weakening of authority have produced marked changes. Looking backwards over several decades in Australia, the reader will say "and we also".

The intimate structure of the individual receives due recognition. The decreasing size of families, the breaks in continuity, divorce, separation and interaction between individuals precede a description of the family cycle. Marriage, child bearing, child rearing, child launching and aging demand a constant readjustment not merely to the individuals concerned, but also to the attitude of the nurse.

Man's life in groups, outside the family, at school, at church, in a profession leads us to the apex of social interaction. The chapter ends with a photograph of the General Assembly in the United Nations' Headquarters. The chapter on "Man Lives in Communities" has interesting sidelights. We are introduced to the implications of subsistence farming, commercial farming, and part-time farming. We learn that small farm communities have numerous advantages. They support more businesses and a higher scale of living.

The author highlights his thesis with case histories of patients; among these is that of a hillbilly from Oklahoma marooned in a large city. Many a patient in one of our capital cities will agree with him: "At home in Oklahoma it was different—because here everybody is out to get his

share and more, and that's the only way you can get it in the city. At home it wasn't like that, because everybody didn't scramble so hard. Here in B—— you have to fight for a bus seat, to get in line at a sale, to do anything. It's kinda dog-eat-dog and you know who takes the fellow that gets left, here in the city. People move so fast in the city that we don't get to know anybody, even the people next door, so I just freeze up and let them go their way and I'll go mine."

As might be expected, race and colour are not forgotten. A sympathetic discussion of the Negro problem has a bearing on that of the Australian aboriginal.

The chapter on "Man Gets a Living" contains information which is surely basic. There are concluding sections on man and his physical health which include an outline of hospital facilities, health insurance opportunities, and most appropriately the effect of illness upon the family. The chapter on man and his mental health stresses the need for prevention commencing in childhood and for efforts to provide psychiatric attention at the onset of symptoms.

A concluding section deals with man's treatment of his social problems. It outlines methods for combating poverty and insecurity. Over the caption that social problems involve more than just people as individuals there is a grim picture of an eroded farm abandoned because of its man-made unproductivity. Facing it is the illustration of a large Federal housing project built to eliminate slums. Such incongruities make the reader "think". Not the least of the book's merits lies in this stimulative quality.

Man cannot escape the impact of his environment, and as this is complex and changing, each one of us, including the nurse, should have more than a nodding acquaintance with the basic principles which are involved. Towards this end, "The Sociology of the Patient" is a valuable handbook.

In the issue of December 4, 1954, a review of "Diseases of the Liver" by Mitchell A. Spellberg was published in this journal. J. and A. Churchill, Limited, of 104 Gloucester Place, London, W.1, have written stating that they are marketing the book in the United Kingdom and Commonwealth countries. The price of the book is £6 sterling.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Aids to Medical Treatment", by T. H. Crozier, B.Sc., M.D., D.P.H., F.R.C.P., and seven contributors; Third Edition; 1954. London: Baillière, Tindall and Cox. 6½" x 4", pp. 542. Price: 12s. 6d.

One of the well-known Students' Aids Series inaugurated in 1876.

"Pediatric Clinics of North America: Symposia on Accidents and Emergencies and Pediatric Allergy": 1954. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 9" x 6", pp. 320, with 37 illustrations. Price: £6 per annum.

This issue is in two parts. The first is a symposium on accidents and emergencies and comprises 11 contributions; there are 16 authors. The second part deals with pediatric allergy; here there are 13 contributions by 14 authors.

"The Medical Clinics of North America": 1954. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. Philadelphia Number. 9" x 6", pp. 304, with 23 illustrations. Price: £7 5s. per year in cloth binding and £6 per year in paper binding.

The whole number is given over to a symposium on cardiovascular diseases, with 18 contributions by 23 contributors. Dr. Wm. D. Stroud writes a foreword.

"Peripheral Nerve Injuries", by the Nerve Injuries Committee of the Medical Research Council, edited by H. J. Seddon; 1954. Privy Council Medical Research Council Special Report Series No. 282. London: Her Majesty's Stationery Office. 10" x 6½", pp. 468, with 276 illustrations, a few in colour. Price: £2 15s.

There are twelve contributors.

The Medical Journal of Australia

SATURDAY, MARCH 5, 1955.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE FIRST WORLD CONFERENCE ON MEDICAL EDUCATION.

The first world conference on medical education was held in London at the end of August, 1953. It was attended by more than 600 persons representing 62 countries and 127 medical faculties. Representatives came from countries so far apart as the United States of America, India, Pakistan, Chile, Peru, Japan and Australia. A simultaneous translation was provided throughout the week in the three official languages of the Conference—English, French and Spanish. Sir Lionel Whitby, Regius Professor of Physic in the University of Cambridge, was President. Readers will remember that the Conference was held under the auspices of the World Medical Association with the collaboration of the World Health Organization, the Council for International Organization of the Medical Sciences and the International Association of Universities. The British Medical Association was the host organization. Dr. T. C. Routley, of Canada, was chairman of the organizing committee and its members were Dr. Hugh Clegg (United Kingdom), Dr. T. Glorieux (Belgium), Dr. Dag Knutson (Sweden), Dr. J. A. Pridham (United Kingdom), Sir Lionel Whitby (United Kingdom), Dr. W. P. Forrest (World Health Organization), Dr. J. L. Troupin (World Health Organization), Dr. H. M. Keyes (International Association of Universities), and Dr. K. Soddy (United Kingdom). Dr. Louis H. Bauer (United States) was Secretary. Dr. Hugh Clegg, of the United Kingdom, was Secretary of the Programme Committee and has also edited the proceedings which have appeared in a handsome volume.¹ It should be stated at once that the production of this volume of proceedings is a great credit to Dr. Clegg, the editor of the *British Medical Journal*, and other members of his staff who have assisted him in the work. To produce a volume such as this and at the same time to continue the publication of the weekly *British Medical*

Journal is an extraordinary feat. Dr. Clegg and his companions had the onerous but fascinating job of designing the style of the book. At the end of his editor's preface, Dr. Clegg refers to the generosity of the Oxford University Press, who left the work of bookmaking in his hands and those of his colleagues and finally gave its expert approval. The volume must be a source of great satisfaction to all who were responsible for it.

The volume, which must be a standby for years to come to all who are interested in medical education, consists of several parts. The first part comprises the opening addresses. Subsequently, there appear four sections devoted respectively to the "Requirements for Entry into Medical Schools", the "Aims and Content of the Medical Curriculum", "Techniques and Methods of Medical Education", "Preventive and Social Medicine"; there are some concluding reports of vice-presidents and rapporteurs. Obviously it is impossible in the space available to give anything like an adequate review of the contents of this volume. The *British Medical Journal* of August 29, 1953, was devoted wholly to the Conference, and several of the more important articles were published in full. Those who read their *British Medical Journal* of that date will certainly wish to purchase a copy of the proceedings. Sir Lionel Whitby, in his opening address, pointed out what we all know, that the world has become so shrunken because of modern communications that we can no longer take a parochial view of our own problems, whether they are economic, social or medical. We do, in fact, live almost in one another's pockets. Sir Lionel Whitby thinks that there is a growing world-wide awareness of the common bonds of medicine and of the contributions which these can make to world peace. What is needed in Australia is a more extensive awareness of these common bonds, especially by those who are engaged in the everyday practice of medicine. They have studied and qualified and entered the ranks of the profession and are free to practise in any part of the world which will accept their qualifications. For many this is quite sufficient. Australia needs to be awakened to her responsibility in the matter of medical education. She needs to ask herself whether she has no contribution of her own to make to the world question. It was singularly unfortunate that none of the three Australian members of the profession who were specially invited to speak at the Conference were able to go, chiefly because no one was willing to provide the necessary finance. Sir Lionel Whitby remarked that just as consideration was given to the ideal medical student, so also it was able to picture the ideal medical teacher. He thought that the selection and appointment of future teachers might well be more deliberate than it had been in order that some might be chosen for their teaching capabilities. He thought that such appointments in some grades might be for limited terms instead of for life. This statement needs to be considered in conjunction with some remarks of Sir Richard Livingstone on education in general when he spoke about the "philosophy of the first-rate". Medical teachers should be chosen from among those who have first-rate minds. Sir Richard Livingstone insisted that three needs determined the aims of education. Education should prepare the student by either a general or vocational training to earn his bread; it should give him some understanding of the universe and

¹ "Proceedings of the First World Conference on Medical Education, London, 1953: Held under the Auspices of the World Medical Association"; 1954. London: Geoffrey Cumberlege, Oxford University Press. 9½" x 6½", pp. 820. Price: 60s.

of men; and it should help him to become a fully developed human being. His remarks were directed chiefly to the last two of these aims; they were the most important to men, but since they were less obvious and more difficult they might be neglected, and especially the last of them. The human being, he insisted, "is a work of art, capable of a quality and beauty of its own, quite apart from any practical purpose to which its powers are put". Sir Richard Livingstone thought that history and literature should enter into any education, for they were the chief records of man and his ways. History was a clinical study of man in society. Literature was more personal. In literature man was met in his intimate moments, talking to the world but really to himself, so that in literature were recorded all the thoughts, feelings, passions, visions and dreams that had ever passed through the human mind, and some of the visions played a great part in human progress. The aim in teaching literature and history to scientists was less to instruct them than to awaken the mind, to make the pupil feel the interest of literature and history, even though his detailed knowledge of them might be small. It was necessary to have some instruction about how to behave when one was introduced into the kingdoms of nature and of man. Several points were mentioned. The first point was to learn how to express oneself intelligibly to others. The second point was to know how to read a book, noting exactly what it said. The third point was to think clearly and logically; logical thinking was one way to truth, but it was not the only road, nor was it a complete one. Another code of behaviour concerned the insight that came from sensitivity, to supplement and correct knowledge which came from hard thinking. Order was not visible in the present world of chaos, nor could a clear voice be heard in the confusion of tongues. Sir Richard Livingstone thought that Christianity would some day become the philosophy of the western world—it was necessary to have a philosophy of life, for without it, it was impossible to think or to express thoughts—but the time was not yet. The only creed which men in every continent in the world could repeat today was: "I believe in science and its child, technology." This was a starting point, it was something; it was not a philosophy. It was at this point that the speaker went on to discuss the "philosophy of the first-rate". He asked how it could be recognized in practice, and answered his own question by stating that there was only one way to get to know it and that was by seeing it, and the seeing of it was the only way in which it could be taught. "It is the same in anything. People learn what is first-rate by contact with it. You learn what is great music by hearing it, what is great architecture by seeing it. You learn first-rate business methods by working in a first-rate business firm. It is the same everywhere. You acquire a taste in good wine by drinking good wine, in good cigars by smoking them."

A perusal of the contribution by Sir Richard Livingstone should be followed by study of the article by Professor John F. Fulton, of Yale University, on the history of medical education.

We must pass to something which will possibly be of more immediate interest to readers of this journal, and that is a contribution by A. L. Richard, Professor of

Obstetrics and Dean of the Faculty of Medicine of the University of Ottawa, Canada. His contribution was entitled "The Undergraduate and General Practice". He tried to answer three questions, as follows: (i) What is a general practitioner? (ii) What type of students do we need if we are to produce general practitioners? (iii) How can the education and training of general practitioners be achieved during the medical course? Professor Richard's first question can be answered by his definition of a general practitioner:

The general practitioner is not a doctor who, having complete, detailed, and systematic knowledge concerning each and every medical discipline, can treat all disorders that may affect the human being. Neither is he to be considered only as a "screener", referring all his cases to specialists. He will be called by the population to give advice and treatment on all and any forms of illness, and, although utilizing the various services offered by the modern hospital, he is, generally speaking, much on his own. He should, then, be ready to cope with any emergency, have adequate knowledge to diagnose and ability to treat the common and ordinary ailments and disorders, and enough honesty, knowledge and common sense to know his limitations and to refer his patients to specialists when necessary. To be able to do this he should be one who, having acquired during his medical course basic professional knowledge and the clinical and social skills necessary to the best utilization of that knowledge, can by habits of reasoned and critical judgment use these principles and judgments wisely in solving problems of health and disease in the community in which he practises. He is one who, possessing and constantly developing these basic intellectual attitudes, has also the ethical and moral principles which are essential if he is to gain the confidence and trust of those whom he treats, the respect of those with whom he works, and the support of the general community in which he lives.

Professor Richard here said all that it was necessary to say. In dealing with his second question, he said that the man who was to become a general practitioner could not be content with being simply a pure scientist with a cold, precise, impersonal attitude to biochemical and physical phenomena. Medicine dealt not of things or events, but with persons. The practitioner had to be a man able to appraise prudently the psychiatric aspect of illness, to understand the emotional complaints of his patient and his reaction to social and personal *milieu*. In a word, in addition to the basic sciences, he had to have knowledge of psychology, of ethics and of sociology; and because he was dealing with persons, he also had to have a sense of values, a philosophy of life. In saying this, Professor Richard backed up Sir Richard Livingstone. In regard to his third question, Professor Richard pointed out that he would be content with indicating only the general lines on which the education of a general practitioner should be pursued. No medical school could expect to prepare only general practitioners, it also had to keep in mind that among its students some would have special qualities or would develop desires to specialize or to go in for research. The basic curriculum should therefore contain the essentials common to both.

It should incorporate general principles of medicine, permitting students, if they do not enter general practice, to have the basic equipment necessary to orientate themselves in specialties or other branches of medicine. The knowledge and training required of a general practitioner will not, later on, hinder a specialist, but will rather be an asset, giving him a sense of proportion and perspective in the exercise of his specialties.

Professor Richard laid emphasis on the subjects which should be taught to enable a student to become a general practitioner of medicine. He did, at the same time, include knowledge necessary to the future specialist. In other

words, he stated the view which has often been expressed in this journal, that the undergraduate curriculum should lay a general foundation on which a student, after qualification, can build his future life work. If he wishes to become a general practitioner, he can use what he has learned of the basic sciences and of their application in everyday medicine; if he wishes to become a specialist, he can use the same principles and the same foundation and build up from that. To hold this view does not mean that no special time is to be given in the undergraduate days to the study of general practice or even to participation in it. It does not mean that a student is not to see and possibly take part in the work of a medical officer of health, investigating foci of infectious disease and so on at first hand. It does not mean that the medical student is not to take an active part in the conduct of a prenatal clinic or in the management of obstetric cases. It does not mean that the medical student is not to follow a surgeon in his diagnosis and assist him at his operative work and later on attend him while he is trying to rehabilitate his patients. It means nothing more than that medicine is an enormous structure which has a common foundation. The edifice which we regard as medicine is a composite affair; it has many rooms and groups of rooms; it may even have towers and turrets, but these are all based on one solid foundation.

There are many other chapters in this book which will well repay study. Mention of them must be left for a future occasion. They are a heterogeneous group and will provide a good deal of scope for discussion and argument. What we have to remember is that there will always be differences of opinion about the teaching of medicine and the place which one or other subject should take in the curriculum. The average lives of men and women are vastly different today from what they were fifty years ago, and the advances that have been made in the basic and other medical sciences have been enormous. We must expect similar changes in the future, both in men and women and in branches of medical knowledge. We may therefore expect that when the second world conference on medical education is held, as it probably will be, in 1958, the views expressed will be different in many respects from those which have been put forward at the first conference. The basic considerations will probably be the same.

Current Comment.

THE ELECTROCARDIOGRAM IN THE EXAMINATION OF THE INDUSTRIAL WORKER.

THE electrocardiograph is finding increasing use in industry in the United States of America as a diagnostic instrument used in preplacement and periodical examinations of industrial workers. It can benefit the individual employee by (a) uncovering disease in an early and possibly remediable stage; (b) identifying benign or inconsequential abnormalities which, without correct diagnosis, may lead to apprehension and anxiety as well as to erroneous employment restrictions; and (c) aiding placement of the worker in a job suitable to his physical limitations if significant disability actually exists. J. R. Durham and L. C. McGee¹ have reviewed 37,000 tracings obtained on

7400 individuals ranging in age from 17 to 75 years. The tracings were made from 1936 to 1952 on workers in a large industrial company in the United States of America. Minor deviations were not considered as abnormalities. Clinical examinations, including laboratory tests and chest X-ray examinations where indicated, were available to aid in the correlation of findings from examinations of the tracings. One hundred and eighty-one persons were found to have electrocardiographic abnormalities classified as left heart strain, myocardial infarction with recovery, right bundle branch block, S-type block, left bundle branch block and auricular fibrillation.

The authors found the electrocardiogram a helpful adjunct in the evaluation of an employee's health and ability to work. However, it must be used in conjunction with other clinical findings. The electrocardiograms were of little help in determining aetiology of heart disease and of no value to prognosis or functional capacity. The records aided appreciably in reassuring the worker with inconsequential signs and symptoms and in obtaining his cooperation for treatment or modification of working and living habits in the presence of potential or actual heart disease. Myocardial infarction is not infrequently an unrecognized episode and has a better prognosis than some hospital statistics indicate. There is no evidence in this study that physical labour or unusual effort predisposed to myocardial infarction or worsened hypertensive disease. Working capacity rests on the clinical evaluation of circulatory function and the worker's training, not on the electrocardiogram. Under proper job placement and satisfactory mental and emotional states the prognosis of the worker with degenerative vascular disease is equal to or better than that of the man who ceases work immediately upon recognition of his disease. Of the group of 51 workers who sustained an infarction and recovered, 14 subsequently have died after working an average of 3.3 years (ranging from five months to eight years) following the first recognized infarction. Of the 37 survivors 22 were still working at the time of the review—an average of just over five years since infarction was diagnosed. From a consideration of their studies the authors set out their ideas as to the economic relations.

The best use of the worker's productive capacity, following the clarification of his cardio-vascular status from time to time, rests largely with others. The physician must be able to define the work attitude and capacity as well as the type of job suited to the individual worker. The employer must have reason, usually from experience, to accept and apply the physician's recommendations on the patient's progress and work capacity. Organized labour must find ways to correlate the rights of the handicapped worker with those of seniority and related considerations in the over-all interests of the union membership. The administrators of compensation laws must learn about and accept the nature of degenerative vascular disease as one basically unrelated to either sporadic or prolonged physical activity per se. . . . Acceptance for employment of workers with known cardiac disease and the continued employment of such men are important matters in view of the prevalence of the various forms of cardio-vascular disease. It seems unwise to load the enormous economic burden of this group as non-productive units onto the backs of the younger workers who are clinically free of disease. Limited physical capacity may mean reduced productivity, and, in turn, reduced income. Is this not better than no economic rehabilitation?

LEPTOSPIROSIS IN THE RICE-FIELDS OF CAMARGUE.

INTEREST in the leptospiroses is perennial, and at the present time a number of investigations into various aspects of their epidemiology and bacteriology are being recorded in this country. Some observations made on a recrudescence of these fevers in Camargue, France, are therefore relevant. Camargue is an island at the mouth of the Rhône, formed by the two main branches of the

¹ *Ann. Int. Med.*, November, 1954.

river, and the reappearance of the leptospiroses is attributed to the fact that extensive and prolific rice-fields have been established there. "M.E.P." comments on investigations made by J. Picard¹ on 100 cases at the *Hôpital d'Arles* from 1951 to 1954. The occurrence of cases has been found to coincide with activity on the rice-fields in the period from May to October—from the planting until the harvest. During this time there are human workers on land that formerly was uninhabited except by animals and migratory birds. The workers have bare legs and arms, which are in direct contact with mud contaminated by the excreta of rodents and animals of all kinds. More men than women are affected. In Picard's series all those affected were either regular or casual rice-field workers, and either of French or of North African nationality.

The particular type of leptospirosis found in Camargue usually has a short incubation period, occasionally only two days. Sometimes the onset is abrupt. In the present series some workers fell ill in the fields as if sun-struck, with headache, stupor, a red and swollen face, and generalized pains in joints and muscles. The temperature rose above 104° F., the pulse rate was rapid, Kernig's sign could be elicited to some degree, the conjunctiva was injected, and the serum albumin content was increased. The clinical picture presented all the characteristics of one of the leptospiroses, at its onset; after a few days the appearance of rapidly fading jaundice clinched the diagnosis. Tests of liver function often revealed transitory alterations.

In Picard's series all types of leptospirosis have been encountered. The meningeal type was characterized by headache, rigidity, and clear cerebro-spinal fluid containing a few lymphocytes, normal amounts of albumin and chlorides, but an increased amount of glucose. The disease might take various intermediate forms between this type and that characterized by severe jaundice and extremely serious hemorrhagic manifestations. Of rare occurrence was a pulmonary form with radiological appearances resembling those of virus pneumonia, clearing in a few days. Attention is drawn to the difference in the clinical course brought about by the introduction of the antibiotics. Previously the disease lasted for about three weeks, divided into two periods of elevated temperature; since the introduction of penicillin there has been a period of high temperature terminating abruptly and permanently. Moreover, the convalescent period has been greatly shortened by penicillin therapy—so much so that the patients have returned to work, and hard work, within ten days to a fortnight; this has made it impossible to carry out the complete investigations that would have been desirable.

From the bacteriological point of view, Picard has for this reason been unable to obtain definite findings. However, in 20% of cases, nearly all of the type characterized by jaundice, he isolated *Leptospira icterohaemorrhagiae*. In the remaining 80% he either obtained negative findings or succeeded in isolating only one of the less virulent leptospires, apparently *canicola*, in very low titre. Madame Kolochine-Eber carried out a great many blood cultures from the patients in Camargue, and isolated an organism which she found to be identical, not with *L. sejrøi* or *L. bataviae* (found in 65% of cases of a similar disease occurring in the Italian rice-fields, especially at Vercelli), but with *L. ballum*. This type was discovered in 1944 by B. Petersen, of Copenhagen, in a mouse (*Mus musculus spicilegus*), and since then it has been isolated from mice only, with one exception—a case resulting from a laboratory infection in the Netherlands in 1948. *L. ballum* is closely allied to *L. canicola*; this would explain the weakly positive findings obtained by Picard.

Some interesting ecological facts are given. In the rice-fields of the Ebro delta, in Spain, the workers are subject to a mild leptospirosis; *L. ballum* has been isolated from their blood. The connexion between the Ebro delta and

the Rhône delta lies in the migration of birds. The zoological station in Camargue, which studies the subject, has proved that waders, flamingos, herons and egrets, which feed on small rodents, migrate from one delta to the other. This is another link in the chain of evidence favouring the *L. ballum* as the causal organism in the fevers of Camargue.

THE USE OF OESTROGENS AFTER THE MENOPAUSE.

THERE are still doubts as to the value of the administration of oestrogens during, but especially following, the female climacteric. E. K. Shelton has discussed the present position particularly in relation to supposed contraindications.¹ The initial purpose of oestrogen production in the female is undoubtedly the preparation of the organism for procreation. This involves all those changes, after the onset of puberty, which make the female attractive to the male. With oestrogen deprivation both the psyche and the soma suffer. There is frequently regression to a shell of the former alluring woman. Sexual misunderstandings may occur and lead to serious family difficulties. The person ages physically and mentally at least in so far as her ego is concerned. Aside from philosophical and sexual aspects of this problem there are several outstanding physical changes which may ensue following oestrogen deprivation.

There are two types of women—physiologically speaking—at the climacteric. One type suffers rather sudden and complete deprivation of oestrogen followed by rapid changes in skin and bone, atrophic changes in the breast and generative organs and severe autonomic instability. The other type may cease to menstruate, but, as shown by good vaginal cornification, continues to elaborate sub-menstrual levels of oestrogens for an additional ten or fifteen years. In the latter the symptoms of oestrogen deprivation occur much more insidiously and may not become grossly apparent until the sixth decade of life. This is why many physicians feel that all women who complain are vapid and neurotic—because they see other women who are practically devoid of symptoms of the menopause. Oestrogen substitution therapy can be of great help to many women of the first class. In the past fifty years the life expectancy of the human female has increased from 48.7 to 72.4 years. Formerly most women who survived into the fifth and sixth decades expected to be, and actually were, senile in both appearance and perspective. By reason of freedom from crippling disease, better nutrition, clever use of cosmetics, greater social freedom and smarter raiment the grandmother of today can hold her place with women even twenty years her junior—provided she remains in some degree physiologically intact.

What are the supposed contraindications for the use of oestrogen substitution therapy? First, the fear of cancer, especially of the breast and genital tract; second, the fear of producing post-menopausal uterine bleeding; third, the fear of prolonging the physiological adjustment to the menopause until the fifth or sixth decade; fourth, the feeling that the menopause is a natural phenomenon and should not be tampered with. In spite of much that has been written there is no sound evidence that oestrogen or any other hormone initiates, generates or produces cancer. A consideration of the amount of oestrogen used in treatment and of the incidence of cancer in the human female supports this statement. Seven hundred kilograms of oestrogens are manufactured in or imported into the United States of America every year. If one milligramme of oestrone is considered the unit dosage, this amount would be enough to treat 7,000,000 women twice a week with one milligramme of oestrogen. If oestrogen was carcinogenic the incidence of malignancy of the breast and sex organs would have been much greater in 1950 than in 1930 when few if any women took oestrogen. Yet

¹ *Presse méd.*, November 27, 1954.

² *Informations méd. SNCF*, July-August, 1954; *Concours méd.*, November 6, 1954.

³ *J. Am. Geriatrics Soc.*, October, 1954.

the death rate was essentially the same in both years. In regard to endometrial hypoplasia women are susceptible to oestrogenic preparations in varying degree, and the dose, preferably by mouth, for any woman past the menopause should be below the level likely to cause bleeding. This may be gauged by experiment. The argument that one prolongs, or rather postpones, the physiological adjustment to the menopause by oestrogen administration is usually answered by the women themselves. Most of them are happy to be relieved of the burden of menstruation and the fear of a late and unwanted pregnancy. There is nothing wrong with having the objective evidence of deterioration in the sixties instead of in the forties. The final argument, that of tampering with nature, can have no validity today with all the interferences that one accepts as progress. The use of oestrogens as a physiological and gerontological tool is an individual and not a general problem. Every patient must be examined and followed and treated as an individual, and suitable patients (and their number is legion) will profit by oestrogen administration.

A BARBITURATE ANTAGONIST.

MANY medical practitioners have been called upon to treat patients who, from misadventure or with suicidal intent, have taken overdoses of barbiturate. In Martindale's "Extra Pharmacopoeia" the remedies against such an occasion are stated to include picrotoxin, nikethamide and amphetamine. T. A. B. Harris has reported an investigation into a new barbiturate antagonist which was announced by F. H. Shaw *et alii* in 1954. This agent is β , β -methyl-ethyl glutarimide. For short, this preparation has been known as NP13. Shaw and his collaborators found that in rats, mice and rabbits NP13, in a dosage of 15 milligrammes per kilogram of body weight, antagonized pentobarbitone, thiopentone and barbitalone anaesthesia, reducing the sleeping time by a half and doubling the barbiturate-depressed respiration rate. In unanaesthetized animals NP13 produced fasciculations and/or generalized convulsions. With doses of 30 milligrammes per kilogram of body weight fatal convulsions occurred in these animals. The intravenous injection of a barbiturate in appropriate dose before or after the injection of NP13 prevented or abolished these convulsions. Shaw *et alii* are stated to have in the Press a report of 39 cases in which suicide was attempted by the taking of barbiturates; a mixture of NP13 and 2-4-diamino-5-phenylthiazole, a morphine antagonist, slightly antagonistic to barbiturates, shortened significantly the period of coma.

Harris was supplied with quantities of these two preparations by Professor Shaw and he used them in three different groups of cases. The first group consisted of 57 psychiatric patients who were given electric shock therapy. In 54 of these cases, NP13 was given and it was found that thiopentone anaesthesia was rapidly terminated without fasciculations or convulsions. (The NP13 was, of course, given after the shock had been administered and the clonic convulsions had subsided, and also after the subject had been decuricized with neostigmine.) In the second group, six patients who had been anesthetized with thiopentone and atropine for orthopaedic manipulations were given NP13. Recovery from anaesthesia was caused by NP13 and the patient fell into a natural sleep. The third group consisted of ten surgical patients who received hyoscine hydrobromide and "Omnopon" premedication and were anesthetized with thiopentone and cyclopropane, together with appropriate *d*-tubocurarine. These subjects had surgical operations lasting from ten to fifteen minutes. The action of NP13 was rapid, though not so rapid as in the second group. In one case among these ten patients, NP13 produced no observable response because the patient suffered from shock and post-prostatectomy clot retention. The failure is put down to the cyclopropane anaesthesia and/or surgical shock. Harris remarks that he has never seen fasciculations or other side reactions when using

doses of 50 milligrammes of NP13. He states that when his results are considered together with those of Shaw, it is justifiable to conclude that NP13 is a barbiturate antagonist of real clinical worth. He thinks that to omit to use it in the treatment of barbiturate poisoning is to run the risk of the bronchopneumonia which often occurs in this condition. Its use after surgical anaesthesia can be valuable, but Harris states that it is more limited because the patient may be under the influence of central depressants other than the barbiturates.

AN AUSTRALIAN ANTHROPOLOGICAL EXPEDITION TO NEW GUINEA.

It has been announced by Sir John Medley, Chairman of the Nuffield Foundation Australian Advisory Committee, that the Trustees of the Foundation in London have made a grant of £4000 to enable an Australian anthropological expedition to travel to the highlands of New Guinea to work there for a year. The expedition will be under the control of the University of Sydney and of the New South Wales Blood Transfusion Service of the Australian Red Cross Society. The personnel will come from the departments of anatomy and anthropology in the university, and is experienced in both the physical and social aspects of anthropology. The Professor of Anthropology, Professor A. P. Elkin, and the Reader in Anatomy, Dr. N. W. G. Macintosh, will travel to the highlands, choose the sites, make the necessary arrangements for the expedition and start the work before returning to Sydney. Specialists in the various aspects of physical and social anthropology, including linguistics, will then carry on the investigations. Specimens of blood will be taken from as many natives as possible and dispatched by air to the Blood Transfusion Service in Sydney. This laboratory has had extensive experience in examining blood from the native races of the Western Pacific Area.

The high country in New Guinea is one of the few areas in the world where native races are still living in almost complete isolation, an isolation which cannot last much longer. Practically nothing is known of their diseases, their physical and social anthropology and their ethnological relations to the lowland natives. The introduction of many infectious diseases into a virgin population is known to be fraught with danger. Quite apart from the scientific interest of the diseases present in such isolated communities, it would be of value to know as many as possible of the diseases which must be guarded against. The difficulties and expense of establishing and maintaining a properly equipped laboratory in such extremely rugged terrain would be so great as to be prohibitive. It would be possible, however, to bleed the natives, send their blood to Australia by air transport and examine it in Australia by modern methods for the presence of antibodies as evidence of existing disease in the area. Modern work on the proteins of the blood has opened new fields of investigation, and already a striking abnormality in the blood proteins of the New Guinea natives has been disclosed. Its significance is unknown, and will be the subject of study. At the same time the physical and social anthropology of the highland natives will be studied and compared with that of other native races of New Guinea. Further, the availability of the natives' blood will make it possible to throw light on their ancestry. This will be done by ascertaining their blood groups in a laboratory in Sydney, a method which is now an indispensable part of physical anthropology. Readers of the journal will be familiar with the important work along these lines carried out by Simmons, Graydon, Semple and others and reported from time to time from the Commonwealth Serum Laboratories in Melbourne.

These races present a unique opportunity to study the sociology, the ethnology and the epidemiology of a native people shut away from the rest of the world by mountain barriers, still accessible only by aeroplane, and it is gratifying to know that the Nuffield Foundation has made possible this important research project.

Abstracts from Medical Literature.

PHYSIOLOGY.

Depressant Effect of Ether on Myocardium.

W. R. BREWSTER, JUNIOR, J. P. ISAACS AND T. WAINO-ANDERSEN (*Am. J. Physiol.*, December, 1953) report that evidence has been presented to show that diethyl ether exerts a direct depressant (negative inotropic) effect upon the myocardium of the dog, which is quantitatively sufficient, in the absence of circulating epinephrine and nor-epinephrine, to produce either a substantial decrease in cardiac output or cardiac arrest at blood ether concentrations required for surgical anesthesia. A major factor in the safety of ether anesthesia, in so far as the effect of diethyl ether upon the myocardium is concerned, is the quantitative reflex release from the adrenal medulla and sympathetic nerve endings of epinephrine and nor-epinephrine, which, by virtue of their positive inotropic effect upon the myocardium, antagonize the myocardial depression of diethyl ether.

Effects of Division of Splanchnic Nerve on Renal Excretion of Electrolytes.

S. A. KAPLAN, C. D. WEST AND S. J. FOMAN (*Am. J. Physiol.*, December, 1953) report that in unanesthetized and anesthetized dogs unilateral division of the splanchnic nerve resulted in a difference in the rates of excretion of sodium, chloride and water and the filtration rate between the two kidneys. The difference was a consequence partly of an increase of excretion of these substances on the operated side as well as of a decrease on the intact side. Evidence is presented which is consistent with the hypothesis that after splanchnicotomy there is loss of tone on the splanchnicotomized side and increase on the intact side. The increase of tone on the intact side seems to be related to the reflex mechanism of blood pressure homeostasis initiated by severance of the splanchnic nerve.

Eosinophile Cell Rhythm in Mice.

F. HALBERG, M. B. VISSCHER AND J. J. BITTNER (*Am. J. Physiol.*, July, 1953) report that mice kept on a regimen providing for (a) *ad libitum* feeding, (b) single housing, (c) controlled environmental temperature ($78^{\circ} \pm 2^{\circ} \text{F.}$) and (d) light during the hours of daytime and darkness during the night show a morning "high" and an early night "low" in number of circulating tail-blood eosinophile cells. When independent groups of mature mice with no history of handling other than that associated with routine feeding and cleaning are examined at these two periods of the day, the early night count is on the average one-third of the morning count. This phenomenon of cyclic eosinopenia is noted in the absence of all recognizable extraordinary stimulation for groups of mice from nine inbred strains and from several stocks of

hybrids, as well as from genetically far less homogeneous populations of mice. Cyclic eosinopenia characterizes mice of both sexes, ranging in age from six weeks to twenty-five months. Its amplitude appears to be smaller for older mice. The temporal placement of cyclic eosinopenia within the twenty-four-hour period in mice fed *ad libitum* is critically dependent upon the schedule of lighting, other conditions being constant. Reversal of the lighting regimen reverses the eosinophile cell rhythm. The reversal of eosinophile cell rhythm by reversal of the lighting regimen is not accomplished at two and at three days. It is slight at four and five days. It is apparently complete in nine days. Reversal of eosinophile cell rhythm by reversal of lighting occurs regardless of the availability or the unavailability during certain periods of the day of a diet not restricted in calories. For mice severely restricted in calories the time of feeding assumes a more critical role than lighting. The alteration of eosinophile cell rhythm by the change in feeding time in mice restricted in calories, and kept on an unchanged lighting regimen, occurs more slowly than the reversal of eosinophile cell rhythm by lighting in mice fed *ad libitum*. The eosinophile cell rhythm is not detected at the usual sampling times after exposure to continuous light for nine days, regardless of (a) the availability or unavailability during certain periods of the day of a diet not restricted in calories, and of (b) the return to continuous darkness for nine days, after previous exposure to continuous light for an equal period. Mice returned from continuous light to a standard alternating light-dark regimen for nine days reveal the eosinophile cell rhythm with its usual temporal placement within the twenty-four-hour period. Mice kept in continuous darkness up to thirteen days, after previous exposure to alternating light-dark for nine days, reveal marked cyclic eosinopenia. The observed facts concerning the effects of environmental factors appear to justify the assumption of an intrinsic mechanism through which environmental effects like those of the lighting regimen are mediated and modified. Data from extirpation experiments suggest that the adrenal gland is probably a part of the intrinsic mechanism of the eosinophile cell rhythm.

Effect of Heat and Hydropenia on Thirst and Chloride Regulation.

G. S. KANTER (*Am. J. Physiol.*, July, 1953) reports that the kidneys of dogs in the hot room conserve both salt and water. There is no significant difference in urine flow in the hot room with or without access to water; and, indeed, while a dog is in negative weight balance an administered load of water does not stimulate diuresis unless that load exceeds the deficit. The urine chloride concentration remains below plasma chloride concentration while the dog is in the hot room with or without access to water. Dehydration up to 10% of body weight has no appreciable effect on urine flow or on the low urine chloride concentration. The low urine chloride concentration is not due to dehydration, for heat *per se* has a depressing effect on chloride

excretion. High environmental temperature does not limit the ability of the kidneys to concentrate exogenous chloride. The urine chloride concentration also rises when the dog is removed to a cool environment. Drinking plays a major role in the regulation of imbalance in the hot room, for here the addition of water alone usually suffices to restore balance in a rapid manner. Actually, dogs with water available *ad libitum* fail to maintain control body weight while in the hot room, owing to failure to drink enough water during exposure to heat. The drinking pattern has individual characteristics. Some dogs take fewer large drafts, other many small drafts; however, the total intake is dependent on the nature of the experiment and not on the drinking pattern. During short runs the total drink is similar, whether taken *ad libitum* or taken after the run is completed; while the drink is greater when taken after a run after a longer period of restriction than if water *ad libitum* is allowed. Short periods of rapid dehydration affect primarily the extracellular compartment, while longer periods affect both intracellular and extracellular spaces.

Modification of Apneusis by Afferent Vagal Stimulation.

D. I. B. KERR, C. W. DUNLOP, E. D. BEST AND J. A. MULLNER (*Am. J. Physiol.*, March, 1954) have described apneustic breathing consisting of alternating periods of apnoea and apneusis in vagotomized rabbits after decerebration in the upper rhombencephalon. The relative duration of the phases depended on the level of section. Eupnoea appeared at times superimposed on apneusis. A type of eupnoea was restarted in previously apneustic rabbits by section of the brain stem just below the acoustic tubercles. Low frequency afferent vagal stimulation during apneusis prolonged apneusis. Medium frequencies of stimulation produced rhythmic respiration at mid-positional levels. Higher frequencies produced sustained rhythmic respiration for the duration of stimulation. After vagal inhibitory influx the next apneusis was greatly prolonged. Mid-line ablation of the upper rhombencephalon slowed apneusis and the eupnoea induced by vagal stimulation during apneusis. It is suggested that the supramedullary regions are responsible for both apneusis and apnoea seen in these experiments. It is proposed that the vagus acts at this supramedullary level. The apneustic centre is assigned to a region at the entry of the eighth nerves, just lateral to the mid-line, and is considered to be possibly some part of the vestibular system.

Effect of Small Infusions of Dextran Solutions.

R. E. SEMPLE (*Am. J. Physiol.*, January, 1954) reports that four clinical dextran solutions that varied greatly in physical characteristics were infused into normal and splenectomized dogs and into guinea-pigs. The amounts of dextran lost from the circulation during the first few hours after an infusion and the amounts appearing in the urine were related inversely to the mean molecular weight of the material infused. From about

six hours after completion of an infusion to the point in time when dextran was no longer detectable in blood, all dextrans left the circulation at about the same rate. It is suggested that this final disappearance rate is related to dextran metabolism. The various dextrans did not differ in their effects on plasma and red cell volumes, plasma concentrations of protein, non-protein nitrogen, sodium and potassium, or hemoglobin concentration of red cells. Plasma volumes were increased by an amount somewhat less than the volume of the infusion and remained greater than control volumes for more than one hour but less than six hours. Circulating red cell volumes were not apparently affected, and the hemoglobin concentration of the cells immediately after infusion was not significantly different from the control concentration. Total circulating protein was reduced as a result of dextran infusions, but the reduction was not significant when the protein removed in blood samples was taken into consideration. Total albumin and fibrinogen were unaffected. Total globulins may have been slightly reduced immediately after infusion, but control amounts were circulating six hours later. Total potassium and non-protein nitrogen in dog plasma were both increased by 15% immediately after infusion, but were within control range one hour later.

Cardiac Variations in Venous Return.

G. A. BRECHER (*Am. J. Physiol.*, March, 1954) reports that cardiac factors influencing venous return have been studied in dog experiments with a new bristle flowmeter of high fidelity. Directly measured blood flow in the superior vena cava is greatly accelerated during ventricular systole when the tricuspid valves are closed. Simultaneously recorded blood pressure and flow curves indicate that this acceleration is caused by the contraction of the myocardium, which expands the atrium through a piston-like descent of the atrio-ventricular junction. Thus, each ventricular contraction not only ejects blood into the arteries but also draws blood from the veins into the atrium. Venous return is only slightly, if at all, accelerated during ventricular diastole. Conclusive experimental evidence is furnished supporting the view that venous return is augmented during ventricular diastole by the direct sucking action of increased negative intrathoracic pressure on the walls of the atrio-ventricular cavity. During atrial contraction flow in the central vein is briefly stopped or even reversed. In the open chest this flow reversal is accentuated by positive pressure lung inflation. The total amount of superior vena caval flow per unit time remains constant at different heart rates. With bradycardia the larger portion of blood enters the atrium during ventricular diastole, whereas with tachycardia 80% of the atrial inflow occurs during ventricular systole. Thus, the heart possesses a self-regulatory mechanism by shifting its atrial filling from a largely passive inflow during the long diastole at slow heart rates to an active systolic attraction of venous blood with tachycardia. It is emphasized that through

this fundamental mechanism the heart assures its own venous return upon which the maintenance of cardiac output depends.

BIOCHEMISTRY.

Testosterone.

H. H. WOTIZ *et alii* (*J. Biol. Chem.*, July, 1954) have shown that a variety of malignant and non-malignant human tissues metabolize testosterone, forming androstenedione as a major metabolite. Normal skin and malignant breast tissues metabolized far greater amounts of testosterone than did any of the other tissues. Prostatic and testicular tissues were able to reduce androstenedione to testosterone.

Cholesterol.

E. J. LANDON AND D. M. GREENBERG (*J. Biol. Chem.*, August, 1954) have studied the dynamic endogenous metabolism of cholesterol *in vivo* by measuring the disappearance of previously labelled tissue cholesterol in rats maintained on a diet free of cholesterol. Important sites of cholesterol synthesis are liver, intestine and perhaps skin. The large scale metabolism of cholesterol must be largely confined to some of the tissues with a maximal turnover rate. These are liver, intestine and adrenal. Because of its extremely high cholesterol content the skin may also play a significant role. The turnover time of liver cholesterol was calculated to be about forty hours. It is suggested that the cholesterol of the testis, spleen, kidney and lung is primarily derived from the plasma. This also appears true for adrenal cholesterol, and its turnover time is equal to or shorter than that of the liver. Castration results in an apparent slowing of the turnover time in liver and intestine. The most significant fact obtained in this work is that the endogenous cholesterol metabolism is far greater in scope than heretofore reported.

M. W. BRIGGS *et alii* (*Arch. Biochem.*, July, 1954) have demonstrated that tritium-labelled sterol appears in the free and total cholesterol pools of the serum of the rabbit after tritium-labelled Δ^7 -cholesterol feeding in a manner qualitatively similar to that following tritium-labelled cholesterol feedings. Tritium-labelled cholesterol was isolated from the liver sterols of rabbits fed tritium-labelled Δ^7 -cholesterol. When this latter compound is fed to rabbits there is produced a rise in the levels of various classes of low density serum lipoproteins. The lipoprotein "pattern" is qualitatively the same as that observed after feeding cholesterol. Feeding with Δ^7 -cholesterol produces a striking rise in serum cholesterol level, and about the same levels are reached as are observed on feeding cholesterol itself. The sterol levels of the serum after this feeding show a maximum of about 12% of Δ^7 -cholesterol. Cholesterol and Δ^7 -cholesterol produce an additive effect on the levels of serum lipoproteins when fed simultaneously. Of the serum sterols 5% is Δ^7 -cholesterol at the end of one week of feeding both this sterol and cholesterol. At the end of two weeks the Δ^7 -cholesterol

level falls almost to zero in spite of continued feeding of both sterols.

Plasma.

H. HOCK AND A. CHANUTIN (*J. Biol. Chem.*, August, 1954) have studied by electrophoresis the changes in plasma and serum of proteins during storage at room temperature. A portion of the β -globulin comprising between 25% and 50% of the total β -globulin increased its mobility within thirty-six hours of storage and was distinguishable as a new component in the pattern. In plasma stored in sodium citrate alone for eight years the changes in the patterns were relatively small, as contrasted with those in plasma stored in the presence of 1% of glucose for shorter periods of time.

Amino Acids.

S. MILLER *et alii* (*J. Biol. Chem.*, August, 1954) have analysed twenty-four-hour urine samples from non-pregnant, pregnant and lactating women for the "free" amino acids, leucine, isoleucine, valine, histidine, lysine, methionine, phenylalanine, arginine, threonine and tryptophane. The median value for each amino acid increased from the non-pregnant to the pregnant state, threonine as much as 350% and arginine only 6%. Increased excretion occurred by the second month of gestation. The quantities of amino acids excreted fluctuated throughout the gestation period, but the median value for each amino acid was always higher than that during non-pregnancy. The median excretion for each amino acid decreased from pregnancy to lactation. During lactation, the median was for threonine only 7%, for lysine 9%, for histidine 12% and for tryptophane 21% of the median during the ninth month of gestation. Each of the amino acids was excreted in smaller amounts than during non-pregnancy, ranging from 50% to 70%. Threonine, histidine, lysine and tryptophane showed the greatest changes during transitions from one phase of the reproductive cycle to another.

Trace Elements.

R. M. FORBES *et alii* (*J. Biol. Chem.*, August, 1954) have reported on the concentration of beryllium, boron, cobalt and mercury in the tissues of the body of a normal adult human, forty-six years of age. Lung, kidney and liver contained the most beryllium (namely, 0.0004 to 0.0002 part per million of fresh tissue), while none was detected in skeletal or voluntary muscle tissue. Maximal concentration of boron was in the skeleton (namely 0.90 part per million) followed by kidney (0.248 part per million). All tissues analysed contained measurable amounts of boron as well as of cobalt and mercury. Liver and skin contained 0.056 and 0.050 part per million of cobalt, with lesser amounts in other tissues, while the greatest concentration of mercury was in the nervous system and heart (namely 0.047 and 0.052 part per million respectively). The concentration of these elements in the tissues represents only a very small percentage of the amounts calculated to be physiologically hazardous.

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on October 19, 1954, the medical superintendent, Dr. NORMAN ROSE, in the chair. The case was discussed by Dr. K. B. NOAD, an honorary physician on the staff of the hospital. PROFESSOR R. McWHIRTER, Professor of Medical Radiology in the University of Edinburgh and Director of the Radiotherapy Department, Royal Infirmary, Edinburgh, contributed to the discussion.

Clinical History.

The following clinical history was presented.

The patient was a thirty-seven-year-old iron-worker who had suffered from "blackouts" for the past two years. The first attack occurred when he was driving a car. He noticed pain in the temporal region and had time to stop the car before losing consciousness for five minutes, after which he recovered completely. Full medical investigation followed, including air studies of the brain, but no abnormality was revealed. The second attack occurred one year later whilst he was working near a furnace. This time he was unconscious for two hours. A third attack occurred three weeks before he was admitted to Sydney Hospital. This time he noticed a peculiar smell as well as temporal headache before losing consciousness for five minutes. There was no reliable witness for any of his attacks, but he had passed no urine or faeces, and had suffered no injury during the attacks.

There were no other symptoms. His family described vague personality changes in the past two years. There were no relevant previous illnesses. There was no family history of epilepsy, diabetes or hypertension. He admitted to drinking alcohol, but moderately. There was no history of head injury.

Physical examination of the patient revealed no abnormality except in the central nervous system, where he was found to have bilateral papilloedema and a doubtful right upgoing toe. The blood pressure was 140 millimetres of mercury, systolic, and 90 millimetres, diastolic. Examination of the urine showed no abnormality.

The day after he was admitted to hospital he suffered an epileptic fit in which he passed through tonic and clonic phases with no localizing features.

The problem was then one of diagnosis and management.

Clinical Discussion.

DR. K. B. NOAD: I was very pleased on reading the protocol, because I thought that this was a patient I had sent in to hospital; and, of course, as diagnosis is always the difficulty in a clinico-pathological conference, I thought that this would be very easy. But some doubt has been cast on that by my surgical colleague. In any case, I do not think there is any doubt about diagnosis, because if we read to the end we must come to the conclusion that this patient had an intracranial space-occupying lesion. Anyway, I am not very worried about beginning this presentation, for after all I am only the overture to the main event on the programme.

The case history of this man presents a problem that we have to deal with every day—perhaps not every day, but certainly every week, either in private practice, hospital or other type of practice, and it is one that gives us very great difficulty and sometimes great anxiety. Now, when we have to deal with disturbances of consciousness, we naturally think of the two main divisions of attacks: the idiopathic attack, such as that type of disorder which, with all its protean manifestations, comes under the heading of idiopathic epilepsy; then the other division, which comes under the heading of symptomatic epilepsy. Those are the two main headings which immediately present themselves to us when we hear a case history like this. To deal with the symptomatic first, I think in the present day tumour undoubtedly takes pride of place. Brain tumour is one of the commonest growths that we have to deal with today. There is another very common cause that we have to deal with today—that is, one of the disorders associated with hypertension. Another is trauma—post-traumatic disorders. Lues has lost its place in this category because it is a disappearing disease. There is another, if I can put it here, and that is alcohol. Alcohol still has plenty of adherents and in some instances is a precipitating factor in convulsive attacks or attacks associated with disturbances of consciousness. Infections are rare, such as encephalitis, but when a

patient comes to the clinic or comes to consultation in private practice, one should always think of metabolic disorders. I always think that, ideally, every patient should have a glucose tolerance test and an estimation of serum calcium.

We can exclude many of these conditions in this instance. Hypertension we can exclude because the blood pressure was 140 millimetres of mercury, systolic, and 90 millimetres, diastolic, and the urine was clear; there was also no suggestive history of any sort. He had been thoroughly investigated in the hospital, so presumably the results of serological tests were negative in the blood and in the cerebro-spinal fluid. He drank but moderately, he said, so I think that we can exclude alcoholism. The history extended over two years, so we can exclude an infective basis. We have no definite information that metabolic disorders have been excluded, but for the sake of argument we will exclude them because of the evidence in favour of tumour later on. Now I kept the question of idiopathic epilepsy to the last. You could say that this man might have had idiopathic epilepsy. After the first attack he was brought into hospital and thoroughly investigated, and no abnormality was revealed. No doubt the clinicians asked themselves whether this could be idiopathic epilepsy. Well, this is a very dangerous diagnosis to entertain for a man of thirty-seven years of age—that is, thirty-five years when the story came on. It is a very dangerous diagnosis to entertain, and one which is made only when one is driven to it. In some figures produced by Aldren Turner from a survey of 1000 cases of idiopathic epilepsy I think that the age incidence went something like this: nil to five years of age, 17% of cases; six to ten years, 14%; eleven to sixteen years, 28%; seventeen to twenty-one years, 18%; thirty to thirty-five years, 3%. Take these figures in another way. Of the 1000 patients only 17% were twenty-four years of age or older; 83% were under twenty-four years of age. So you can see the risk one takes in making the diagnosis, or even entertaining the diagnosis, of idiopathic epilepsy in a man of thirty-five. It is rather a coincidence that the last patient I saw before I came up here just now brought a letter from her doctor saying that Mrs. So-and-So, aged sixty-one years, had been regarded as having a cerebellar vascular lesion. "But I should like you to see her to make sure that she has not a tumour in view of the fact that her son was under treatment by the Repatriation Department and considered to have idiopathic epilepsy, but he turned out to have a cerebral tumour." That was quite remarkable, seeing that I was coming at once up here to this discussion.

Now are there any features about this protocol which would make one think of tumour, that is, before he developed papilloedema? Yes; did you notice that the second attack, a year after the first, was associated with a period of unconsciousness of two hours' duration? If you have a patient who has any disturbance of consciousness or a convulsive attack followed by a period of unconsciousness of two hours' duration, then that patient has a tumour unless you prove otherwise. Twice he had pain in the temporal region. There is not much in that. After all, we all know that migraine and other neuro-vascular episodes can produce pain in the temporal regions. You will notice, too, that the third attack was associated with a peculiar smell. That is not necessarily diagnostic, but it is an important point to remember. The important thing is the duration of unconsciousness in the second attack. Finally we have the history from the relatives saying that he had vague personality changes in the past two years. These three or four points to my mind are helpful, and together with the statistics I have quoted indicate that the diagnosis in this patient was probably a cerebral tumour.

Now the story extends over a long time, two years, and so it is probably a slowly growing neoplasm. Of course, the slowest of all neoplasms is the meningioma. Another slowly growing infiltrating type of growth is the astrocytoma. It may have been an infiltrating astrocytoma because, even after two years, there was only a questionable upgoing toe on one side. Then there is the oligodendroglioma, which also is a slowly growing neoplasm, but there was no evidence of calcification in the X-ray films such as one might expect with this type of tumour. One must think, then, that this patient had a slowly growing neoplasm, which may have been a meningioma or an infiltrating type of growth in the nature of an astrocytoma. Now I think it is reasonable to use all the evidence available. I am sure we would not be asking Professor McWhirter to discuss a meningioma because our surgical colleagues would, no doubt, have attempted to remove it. So I will say that this patient probably had an astrocytoma. A glioblastoma I think we can exclude because of the long duration of the history. This, however, is not absolute, as a glioblastoma sometimes

exists longer than we think, especially when there is more cellular differentiation than in the usual type of hemorrhagic growth we see.

I was asked to say what investigations would be done when confronted with a problem like this. First and foremost is a very good history, and second is a thorough physical examination. Then we come to our ancillary evidence, like X-ray films of the skull and electroencephalography, and, in consultation with our surgical colleagues, angiography would probably be used in the diagnosis. Any interference with the spinal theca would necessarily be left to the last or not done at all because of the papilloedema which is evident here. It is extremely dangerous, of course, to perform a lumbar puncture, or to do any investigations involving the spinal theca in the presence of papilloedema. So, if I may conclude, I think that this patient had a cerebral tumour probably of an infiltrating kind, probably an astrocytoma.

DR. N. H. ROSE: Before we finish with you, Dr. Noad, we have a distinguished lot of consultants with us here today. Are there any of those whose opinions you would like to ask? Would you care to examine these X-ray films? Dr. Jenkin.

DR. R. JENKIN: The first of these films was a plain X-ray film of the skull showing an enlarged pituitary fossa with destruction of the posterior portion of the fossa, and that is about all. At this stage it is impossible to say whether the destruction was due to an intrasellar or suprasellar lesion. A further set of plain X-ray films was taken about three weeks later. Again they showed the same thing—an enlarged pituitary fossa and some destruction of the posterior part. About a fortnight later a cerebral angiogram was done. It shows good filling of the branches of the internal carotid. The middle cerebral artery shows a displacement upwards and away from the temporal region with an absence of vascular markings in that region. The inference is that there is a lesion in the right temporal zone. It seems to be a fairly avascular tumour. That is as far as we can get.

DR. ROSE: Are there any other opinions you would like, Dr. Noad? Dr. Arthurs.

DR. G. ARTHURS: What I would like to take up first is the point that he had no pain in the temporal region before the first attack of unconsciousness. There was no history of headache and vomiting. Well, quite a lot of tumours that we see give rise to no headache and vomiting. On the other hand, a condition which has not been emphasized very much in the books, but which we see quite a lot of, would fit in very nicely with this history; that is wasting of the brain. It is a condition which gives rise to fits in young adults, and very often one gets caught when about to do air studies as a preliminary to craniotomy by finding a low pressure of cerebro-spinal fluid, and the fluid comes out in bucketfuls. That is the first warning. Then, looking at the air pictures, one finds a wasted brain with big black ventricles, quite symmetrical, with a lot of air on the upper surface. This condition is quite common, but it is not very well written up in the books. Unfortunately, there is very little one can do about it except to treat the symptoms.

Another interesting point was bilateral papilloedema and no localizing signs except for a right upgoing toe. This signifies intracranial hypertension. Most people that we see with intracranial hypertension like this, but with no localizing signs, have one of two things. Either they have a posterior fossa lesion (not necessarily a tumour—sometimes it is an inflammatory thing like tuberculous meningitis, which I have been told you do not see in this country, but we see a lot of in England) or a space-occupying lesion in the temporal lobe. Another point was that he noticed a peculiar smell. Now, I would say that that is quite an important symptom. I should like to have gone into it a little more fully. I would like to know whether he had a feeling of things having happened before, a feeling of unreality, what is called the *déjà vu* phenomenon. Sometimes these people have hallucinations of sight, and, just occasionally, they hear voices; and then they are mistaken for psychopathic cases.

DR. ROSE: Dr. Noad, is there anything further you would like to add to your diagnosis of astrocytoma, having seen the X-ray films? Would you like to localize this lesion?

DR. NOAD: Yes, I would like to say that it is in the temporal lobe, probably on the right side, despite the fact that the upgoing toe is on the same side, because there is no history of aphasia or anything like that.

DR. ROSE: Now, before the diagnosis is revealed we have a lot of all-round people here, general physicians, surgeons,

neurologists, pathologists, haematologists, and we would like them to air their views. Although the diagnosis is fairly apparent, we would like some hints on the localization and on the type of this lesion. Have we one of the members of the medical staff or one of the visitors who would care to hazard a diagnosis? Dr. Nagy, would you care to say something?

DR. G. S. NAGY: The arteriogram suggests a lesion in the right temporal lobe and the symptoms fit in with this. Peculiar smell is very characteristic of temporal lobe lesions. I remember one such patient whose sensations of smell reminded him of the odour of Arab villages in the Middle East. The loss of consciousness for short periods and the personality changes in the present case are further evidence of a lesion in the temporal lobe extending, perhaps, into the parietal lobe. Personality changes are perhaps more common with lesions which extend beyond the temporal lobe.

DR. ROSE: Thank you, Dr. Nagy. Is there no further discussion on the diagnosis? We will ask Dr. Palmer to read out the operative and pathological findings.

Pathology Report.

The following report was given by Dr. A. A. Palmer, and slides were shown.

On May 18, 1954, several fragments of pale grey tissue together measuring 0.7 centimetre in maximum extent and a tissue smear were received. On microscopic examination much of the tissue consisted of moderately cellular tumour with nuclei of variable size, some rather large. Cells resembling gemistocytes were present in places; there was abundant fibrillary background; some neurons remained within the tumour. The appearance was that of a moderately cellular astrocytoma.

On June 1, 1954, the following specimens were received: (i) Labelled cortex: one small and two large pieces of cortex with underlying white matter and a tissue resembling tumour, together weighing 45 grammes; the largest measured 6.5 centimetres in maximum extent. In one place the grey matter was very pale and not clearly demarcated. (ii) Labelled cortex and tumour: numerous pieces of brain with tumour, together weighing 42 grammes. (iii) Labelled tissue tumour: several small pieces of greyish translucent tissue. On microscopic examination tumour was present in six of seven sections examined, including tissue from each of the three specimens. The growth was a moderately cellular astrocytoma as before.

Discussion.

DR. ROSE: Would Dr. Hurt give a brief description of the operative findings?

DR. B. R. M. HURT: On June 1, 1954, craniotomy was performed. The neurosurgeons removed from the right temporal lobe, plus a deep extension into the parietal lobe, a tumour which they said did not extend to the surface.

DR. ROSE: Well, then, the diagnosis made by Dr. Noad and supported by Dr. Nagy was correct. Dr. Duval, would you say a few words on the radiotherapy of the lesion?

DR. F. DUVAL: This was a slowly growing astrocytoma, and the response to radiotherapy of these tumours is usually not good. I think that deep X-ray therapy is justified, either following operation, or if the tumour is inoperable. Sometimes there is a slight effect on the tumour, in that its progress is slowed and the possibility of recurrence is delayed some time. In addition radiotherapy of an inoperable tumour sometimes has a worthwhile effect in lessening vascular supply and thus reducing the amount of the space-occupying lesion. Even in an inoperable case, therefore, there may be some relief of intracranial compression. The dosage has to be quite high, depending on the size of the lesion. In general, we treat well beyond the limits of the tumour. For a wide field we tend to keep the dose down in the region of 4000r to 5000r given in a period of four to five weeks. If the area of the tumour is small the dosage is increased to about 5500r.

It is very difficult to estimate, of course, how much benefit a patient has received, because almost invariably there has been some surgical help before we see the patient, and it is difficult to decide how much of the improvement is due to the surgeon and how much to the radiotherapist. In some cases, and I think this is one of them, there is a gradual improvement during treatment. When this patient was seen about two months later most of his symptoms had disappeared. I think, even if one accepts that response of the tumour is small, it is wise to give the patient the benefit of the doubt and to treat it.

DR. ROSE: This discussion has assumed rather a radiotherapeutic angle. This might be diverting a little from the normal purpose of these meetings, but we so seldom have the opportunity of hearing an authority such as Professor McWhirter on this subject that we asked if he would say a few words.

PROFESSOR R. MCWHIRTER: Thank you, sir, for asking me to take part in the discussion. Most of the remarks I will make are based on the results of an investigation done with Professor Dott in Edinburgh. To begin with we irradiated all brain tumours and then, after a period of time, tried to determine just what benefit had been obtained. In the astrocytoma series we got a survival rate at the end of five years—I am speaking from memory—of something like 47%, if I remember correctly. That seems very satisfactory, and one might easily conclude that radiotherapy had played a very useful part in treatment. It was rather disturbing to take a similar series of cases, so far as could be judged, treated entirely by surgery, without any radiotherapy, and find that the survival rate was not significantly different.

We had, too, an opportunity of investigating tumours where the extent of the tumour was determined, the patient was given radiotherapy, and at a later date the skull was reopened. We were unable to satisfy ourselves that there was any real shrinkage in the tumour. The normal life history of an astrocytoma, especially if decompression has been performed, and where a portion of the tumour has been removed (in this case quite a large portion was removed), can be anything from ten to fifteen years. Even in the absence of operation the life history of some of those tumours can be very long indeed, as shown by their symptoms. These, of course, gradually become more marked as the process extends, but often there is no doubt that the tumour has been present for a very long period of time. Every now and then a slowly growing astrocytoma may develop a much more malignant focus within it, the so-called glioblastoma, and then the progress of the disease is much more rapid. The patients usually die within a year of the onset of a glioblastoma, which may, of course, develop independently of an astrocytoma.

In summary, then, our conclusions have been that we have not really contributed anything by radiotherapy in these cases. We intend trying again when supervoltage becomes available. Not because there is any reason why supervoltage irradiation should produce any real difference in biological effect, but we propose to plan the investigation along somewhat better lines, in that each patient, after operation, will be selected at random for radiotherapy. The random selection can be done by any suitable method; tossing a coin is perfectly satisfactory, though not very professional, or you can look up a table of random numbers, which sounds a lot better. If a patient comes into the category for radiotherapy we propose using supervoltage irradiation. Then we will have two groups of cases selected at random, and it will be possible again to ascertain whether radiotherapy has any effect at all. The conclusions at the moment are that radiotherapy does not contribute anything worth while. Thank you.

DR. ROSE: You have heard the results of a very long experience of the treatment of these tumours with radiotherapy. I think we have some time left. I do not think we have inquired sufficiently carefully into the diagnosis of these conditions. I just wonder whether Dr. Marsh, who is here, would like to say some words on the radiological diagnosis and localization of these tumours.

DR. H. G. MARSH: This is a type of glioma, or glial tumour, and it produces changes which are perhaps best demonstrated by an angiogram. Because this is a widely infiltrating tumour it tends to surround the vessels as well as to displace them. You notice how, as Dr. Jenkin pointed out, the middle cerebral artery is displaced, and you notice how fine these vessels are in the branches of both the middle and anterior cerebral arteries. That is a feature of astrocytoma and other slowly growing tumours. Another thing is that there are no real tumour vessels shown in any of these angiograms. The usual series of angiograms, as you know, involves a picture of filling of the arteries, a picture of filling of the capillary phase of circulation and a picture of filling of the veins. You can see that in this arterial phase there is displacement and narrowing of vessels, and in the capillary and venous phases you note absence of tumour vessels. In the more malignant tumours, the more rapidly growing ones, newly developed tumour vessels are a very prominent feature; a great rash of vessels appears within the tumour area. I think you can say the angiogram is the most useful radiological investigation of this cerebral tumour.

DR. NOAD: Is the pushing-up of the vessels an index of the slow growing of the tumour?

DR. MARSH: I think it is, in a way, although from that picture you could not say what sort of a tumour it is. From the whole series, I think you can say this is very likely a slowly growing glial tumour such as an astrocytoma.

DR. NOAD: What about that pituitary fossa?

DR. MARSH: That is the picture of a pituitary fossa which is deformed by increased intracranial tension going on for a long time. It is not the picture of a fossa which is the seat of a tumour. You notice that the posterior clinoid processes are thin and seem to be vanishing at the top. The sella itself is not greatly enlarged. The essential features are the thinning of the posterior clinoid processes and resorption occurring at the top.

DR. ROSE: This meeting has been clinical, therapeutic and diagnostic so far. We have quite a strong body of pathologists here. Would any of them like to throw any light on the interpretation of these tumours? Dr. Palmer, have you any more to say in discussing this case?

DR. A. A. PALMER: Only that there was no difficulty in making the diagnosis from this little bit taken off the top, but you can get gliosis at the surface of a lesion which is really malignant underneath, and interpretation of a small section taken from the surface of a tumour can be very difficult. It is not always certain whether you are dealing with a glioma or with tissue which has been altered by pressure from some underlying lesion which has not been included in the biopsy material. In this case we were fortunate enough to get a reasonable amount of material in the biopsy, which shows, I think, sufficient variety in nuclear size and sufficient cellularity for us to be reasonably certain that, even with a small piece, it was a glioma, probably an astrocytoma. The other thing is, as Professor McWhirter said, that a tumour of greater malignancy may supervene, and, again, in different areas of the same tumour you may find a very different picture—a much more anaplastic region in one area and a well-differentiated astrocytoma in another.

DR. NOAD: May I ask Dr. Palmer a question, please? Have you ever seen *gliomatosis cerebri*—that is, a cellular infiltration throughout the hemispheres, mid-brain and so forth, without any definite localization into a tumour mass?

DR. PALMER: Yes, I have seen it on one occasion. The brain was unusually enlarged on the one side by diffuse gliomatosis, and the two sides could be compared. It would be much more difficult to diagnose if it affected both sides.

DR. NOAD: Was there anything diagnostic about it clinically, or was it just found *post mortem*?

DR. PALMER: Purely a *post-mortem* finding.

DR. H. M. WHYTE: I should like to ask a question. In the irradiation of tumours in the skull such as this or in other situations within the skull, what is the likelihood of damaging the pituitary? As an extension of that question, how effective is irradiation of the pituitary area, particularly in other conditions such as exophthalmic ophthalmoplegia?

DR. ROSE: I think that is directed towards our learned guest.

PROFESSOR MCWHIRTER: I think the normal pituitary seems to be capable of tolerating as much dosage as the rest of the brain can be given without any change whatsoever. We have irradiated a number of tumours now at the base of the brain where the pituitary would receive full dosage, and it seems that the normal pituitary will tolerate very high dosage indeed. In the presence of tumours of the pituitary itself, a change can be produced. The most dramatic results, of course, are obtained with basophilic cell tumours—Cushing's syndrome—which respond dramatically; and so, too, in the acidophilic group a good, though less dramatic, response can be obtained. A portion of the chromophobe tumours shows a response to irradiation.

In exophthalmic ophthalmoplegia we have been running two series, irradiating the pituitary in one, and in the other irradiating the posterior part of the orbit. On the whole the patients with irradiation of the posterior part of the orbit have been doing better, contrary to what we expected. The basis of treatment was to reduce thyrotrophic hormone, and we thought that if the pituitary were not included, little benefit would be obtained on the rather oedematous and fatty tissue in the posterior part of the orbit. It is difficult, of course, to make quite sure that you irradiate only the posterior part of the orbit, or irradiate only the pituitary—they are so close to one another. I do not think it is surprising that the pituitary should be sensitive when it is abnormal. The same thing happens with the thyroid.

A normal thyroid will tolerate as much dosage as can safely be given without causing myxœdema. On the other hand, an abnormal thyroid, as in hyperthyroidism, can be affected by radiation, though the effect is by no means dramatic.

DR. ROSE: Dr. Noad, would you care to say a few words in summing up?

DR. NOAD: I am sure we have all enjoyed the discussion. The only other thing I can say is that here again is the problem of a comparatively young man having disturbances of consciousness, and these attacks have been proved to be due to a slowly growing, infiltrating growth of the brain. As I said before, this is often a very difficult diagnosis to make when the results of early investigations are negative, but one must never discard the possibility that there may be a space-occupying lesion responsible for these attacks.

DR. ROSE: Our thanks go to those people who have taken part in the discussion, particularly to Professor McWhirter, who has given to us a whole afternoon of his short time in Sydney.

Diagnosis.

Astrocytoma in the right temporal lobe of the brain.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on November 25, 1954, at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, Dr. T. Y. NELSON, the President, in the chair.

Hospital Records.

DR. J. C. FULTON read a paper entitled "Hospital Records" (see page 337).

DR. H. SELLE said that one point had struck him very forcibly: that was Dr. Fulton's reference to the fact that he considered it the moral responsibility of the honorary medical officer to see that a proper medical history was kept. Dr. Selle did not doubt that there was a moral responsibility; he thought that the stage was rapidly being reached at which it would be a legal responsibility. All those present knew, for example, that if an orthopaedic surgeon who was treating a patient for a possible fracture omitted to arrange for an X-ray photograph of the limb to be taken, he might at law be held to have been negligent. Hospital administrators also had—along with the medical staff—a responsibility to see that proper records were kept. The hospital and the doctor might be joined in a legal action for negligent treatment. Dr. Selle thought that in the not distant future an honorary medical officer might find, as the result of case law, that he had a legal obligation to see that a proper record was kept of the treatment of patients under his care. The point that Dr. Fulton had stressed concerning moral (and one might add possible legal) responsibility was very important.

DR. C. C. MCKELLAR said that he felt almost inclined to apologize for absent colleagues that so few should be present at such a valuable address. Also, it should really be heard by hospital secretaries and hospital board members, who stood much in need of the information and of the stimulus so provided. He went on to say that common honesty in classifying results demanded a heading "worse" or "aggravated" to match the heading "relieved". It was often impossible with honesty to put any result immediately on discharge and the true entry in the result space was then "nil" (for example, at the Royal Prince Alfred Hospital in Dr. McKellar's student days patients had come in with the diagnosis "sterility" and left hospital with the result "cured"). He questioned whether there should not be a delayed review of histories in order to enter a truer "result". He asked what record systems were in vogue at the main teaching hospitals, and in the case of a small hospital just commencing a filing system how much might be lost from being out of step with the others. On the question of how long to keep records, in other words, the ideal period (without immediate regard to available storage space), he felt that the suggested twenty-five years was not enough, but that it should be the lifetime of the patient; for example, in orthopaedic cases it might be valuable to look up the childhood history of a patient presenting himself in middle age with a painful hip.

DR. R. B. WILES referred to the long time for which hospital records might be of value—a much longer time than their legal value. Dr. Wiles said that after a term as junior resident medical officer at the Royal Prince Alfred Hospital he had had the opportunity of acting as a *locum tenens* in a practice where the records were available for seventy years. If one wished to inquire into the question of familial disease or something similar, one could look back to the time of the grandparents or even great-grandparents of the present patients. Many of the families in the practice were old families who had not moved out of the district. Dr. Wiles pointed out that if in future years it was desired to gather any information of value about heredity in association with disease, the destruction of records after six years, fifteen years, or even twenty-five years would eliminate their value to the medical profession for research purposes. Dr. Wiles thought that their aim as doctors should be to find some means of overcoming the necessity for destroying records because there was insufficient space to keep them. He thought that there should be some central record bureau where old records could be kept for future reference, because ultimately medical research would move along those lines. It was tending that way now, but there were insufficient facts. Dr. Wiles also commented on what Dr. McKellar had said about the question of honesty in keeping records. He said that from the legal point of view he realized that honest records could not always be kept. As an example he mentioned an operation at which the pancreas instead of a piece of omentum was sewn over an ulcer. An obstruction followed, and a subsequent operation showed what had been done. Such things could not be explained in the medical records. Dr. Wiles wondered whether Dr. Fulton could suggest some way in which an honest medical record could be kept, apart from the legal dishonesty of records. It was a difficult problem, but from the point of view of medical research the matter was important. Dr. Wiles then said that in his opinion when a diagnosis could not be made, the patient's condition should not be labelled as a particular disease. That had nothing to do with the legal aspect; but if the hospital records were to be used for research purposes, they should be detailed and accurate. An example had occurred at the Sydney Hospital chest clinic. There they had kept, as they thought, detailed and accurate records of about 60 cases of carcinoma of the lung. After several years they wanted to survey their records in order to get out some statistics and details. The records had been kept with that purpose in view; but the survey could not be completed, because the records were quite inadequate. That took one back to the medical school. Professor Lambie had received considerable criticism for the type of record which he said should be kept for each patient—one containing the positive and negative findings and every other detail concerning the patient's medical history, for purposes of medical research or medical reference later on. The result of the attempted piece of clinical research at Sydney Hospital showed that unless such detailed records were kept from the medical point of view, they would be of value for statistics and for legal purposes, but not for medical research. At all the teaching hospitals, at least, the records were of little value for medical research as they were generally kept and maintained at the present time. Some solution to that problem would have to be found if they were going to advance medically.

PROFESSOR LORIMER DODS referred to what Dr. Fulton had said about the necessity for the honorary medical officer to assert at the bottom of a clinical history sheet that it was a true and accurate record of the patient's illness. Professor Dods asked for Dr. Fulton's comments on the suggestion that in appropriate cases the honorary medical officer should write a brief personal comment on the history sheet. He himself had recently been trying to do that, and had found it a stimulating clinico-clerical exercise. Professor Dods had also been interested to hear what Dr. Fulton had said about nurses' records; he wondered whether it would be reasonable to ask the sister in charge of a ward to add some brief comments to the history sheets of occasional sick children.

Dr. Nelson, from the chair, said that some important questions had been raised by the various speakers. Probably the most important point was that of the responsibility that the honorary or visiting medical staff of a hospital must be expected to carry in relation to records. That was inescapable. It was the duty of a physician, not only to give good care to his patient, but also to see that the records were maintained and would be of use. That question affected not only the public hospitals, but also the private wards of those hospitals which had them. It was notorious that the records of the private side of any hospital were not so

good as those of the public side. There were two reasons for that. In many cases a doctor kept his own records, and for that reason the hospital record suffered; but his record was still in the final analysis considered as part of the hospital record, and it undoubtedly should be kept as part of the record that was available to that hospital for its various purposes. All sorts of ramifications had developed in recent years, one of which was an increasing use of photography in accurate record keeping. The plastic surgeons knew its value, and now no record of a plastic procedure was complete without photographs of the condition before and after operation. Dr. Nelson said that one of the important parts of record keeping that had not been mentioned was that every hospital which received patients owed an obligation to the practitioner who sent a patient into hospital. It should be a duty of every hospital to furnish the practitioner who supplied that hospital with clinical material with an adequate record of what had happened to the patient during his stay in hospital. That all added up to a very considerable burden, and one which should be willingly accepted by the staff of the hospital, as just as much part of their duty as giving the patient reasonable medical care. Many other aspects of the subject could be raised, but sufficient had been said, firstly, to show that the ideal stated by Dr. Fulton had not yet been attained, and secondly, to show that it was the duty of those medical men who were associated with hospitals to see that their records were kept better. Dr. Nelson suggested that that applied not only to teaching hospitals, but to any hospitals, including base hospitals, that received patients from practitioners who were not on the staff.

Dr. Fulton, in reply, said that Dr. McKellar had referred to the recording of results of treatment—"relieved", "unrelieved", "died" *et cetera*. There were two schools of thought: (i) that no headings were used at all, as the physicians and nurses merely wrote their comments in chronological sequence; (ii) that a large amount of detail was set out on printed forms. The second method was used in some hospitals as a reminder to the writers not to leave out one or other detail, and not to forget to make a comment on some particular aspect. Dr. Fulton thought that such printed forms were merely guides, and that one was not confined to them at all. The attending physician should honestly record there whatever he thought should be recorded. The second question asked by Dr. McKellar referred to remote results; that was the reason why hospitals maintained records and made them available, because it was the attending physician who followed up remote results. With regard to the length of time for which hospitals should keep records, Dr. Fulton said that he thought they should be kept permanently. Dr. Wiles had also referred to the value of records kept permanently. Dr. Fulton thought that the microfilm method was the answer to that problem. The ordinary film had the same length of life as good rag paper—approximately 100 years. As the bulk of microfilm was small, once the records were beyond their active lives they could be photographed on microfilm (after twenty-five years). Mrs. Huffman considered that records were rarely called for after twenty-five years and seldom after fifteen years; their most active life was up to fifteen years. Dr. Fulton could see no reason why records should not be kept permanently. He thought that hospitals would at present be loath to turn their records over to a central agency. On the question of classification, Dr. Fulton said that it was difficult to say what classification a hospital should adopt. At the Royal Alexandra Hospital for Children they had adopted the Standard Nomenclature only a year or two previously; but when they made the change, they had it in mind that there was no difficulty in changing back to the International or some other nomenclature, except that it would be advisable to make the change at the beginning of a year. Trained medical record librarians could maintain a diagnostic index or operations index coded for the Standard Nomenclature or whatever one was being used, and also for the International Classification; at the back of the International Classification was a key which gave a code for transposing Standard Nomenclature into International Classification. Dr. Fulton said that he would say himself at present that it would be best to adopt the United States Standard Nomenclature because of its fine detail and its adaptability to the requirements of clinicians. However, it had to be kept in mind that the International Classification would probably be much improved in the next few years, and it might be necessary to change over to it, because it was an international one. At present it was more suited for statisticians than for the requirements of clinicians. The Standard Nomenclature dealt with the individual; the International Classification dealt with large groups of individuals, and lost sight of fine detail. On the

subject of out-patient records and their variety, Dr. Fulton said that he thought most specialists liked to have their own particular form of record, and they were provided. Dr. Fulton pointed out that under the unit record system there would not be cards. If the in-patient record was of quarto size, then the out-patient record would also be a quarto sheet. Cards were not used, only paper sheets. At the Royal Alexandra Hospital for Children they had a large number of printed forms for various treatments and for control investigations, and the average medical record consisted of 37 sheets. When it was possible to add out-patient records under the unit record system, which he hoped would be in three or four years, the out-patient record would in his opinion mean the addition of an average of only another three or four paper sheets. Referring to the question of honesty in records, Dr. Fulton said that it went right back to the medical attendant. He thought that in 99% or more of legal claims no man need hesitate to put in everything with complete honesty, because all that information would still be to his benefit. Dr. Fulton's experience had been that the more complete the detail was, the more the court was impressed by its straight-out honesty. The courts seemed very tolerant and understanding on that point. They were more likely to regard a record as an honest one when something was recorded against oneself. Dr. Fulton agreed with Professor Dods that the personal comment was a good idea in the record. When they had achieved having the attending physician sign the record, then they might be able to induce him to add some personal comment. Dr. Fulton said that when he mentioned the nurses' records, he was referring to the graphic chart and also to the night and day ward reports. He understood Professor Dods to be asking about the incorporation of the nurse's comments in the actual record. Provided that it was clearly indicated that they were her comments, he thought it would be in order; in some hospitals the nurse's notes were recorded on quarto sheets of a special colour. When a researcher was going through those records, he knew immediately whether he was looking at a nurse's notes or at a doctor's notes. Dr. Fulton thought there was some advantage in keeping nurses' notes separate from doctors' notes. Dr. Nelson had mentioned private records. Dr. Fulton said that it was desirable that in a hospital private records should be filed numerically with the public records. He thought that the hospital must keep full details, and that it was entitled to expect the attending physician to prepare them. If he did that, the hospital should be prepared to make a copy of those private records and supply it to the physician for his own files. That was one suggested way out of the present difficulty. At present private records were not good records, and from the legal point of view medical attendants on private patients who were not keeping accurate records in their rooms or in the hospital were putting the patients and themselves in jeopardy. Dr. Nelson had mentioned clinical photographic records. They would obviously become a part of the hospital record. On the question of referring practitioners being sent a full account of the patient during his stay in hospital, Dr. Fulton said that there was everything to recommend that. In Chicago he had heard an interesting statement of what good public relations were: "giving good service and getting credit for it". Sending a detailed account to the patient's own doctor of what had been done for the patient in hospital would increase good public relations.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

SYDNEY HOSPITAL.¹

[From "The History of Sydney Hospital", by J. F. Watson, M.B., Ch.M., Sydney, 1911.]

PROPOSAL for erecting a General Hospital in the town of Sydney and a wall surrounding the same, by Messrs. Blaxcell and Riley,² in conformity to the plan and dimensions of His Excellency Governor Macquarie.

¹ From the original in the Mitchell Library, Sydney.

That as remuneration for erecting the aforesaid Buildings and finding all Materials for the same the Contractors submit the following propositions to the Sanction of His Excellency, viz.:

1st. The Building to be commenced on the 1st day of May 1811 and to be finished⁽¹⁾ in three years from that period.

2nd. The contractors to be allowed to purchase from the first vessel importing Spirits 4,000 Gallons, for which they are not to pay a greater price than the Government pays for such proportion of Spirits as they may engage.

3rd. The duty of 3s. per Gallon on the Spirits thus purchased to be paid in six months from the date of its being landed in the Colony.

4th. To be allowed the use of twenty draught bullocks from the Government Herds which the Contractors will engage to return at the completion of the buildings, or make good any loss that may happen to the Bullocks by Death or accident in that period.

5th. To be allowed on their Arrival to take off the Stores and to maintain and cloath free of any expence to the Crown Twenty male convicts for the term of three years.⁽²⁾

6th. To be allowed permission during the erection of the Buildings either to import from India or to purchase in the Colony 41,000 Gallons of Spirits exclusive of the quantity mentioned in the 2nd. article of these Proposals with the same Credit for payment of the duty as specified in the 3rd. article.

7th. To be allowed to vend to the best advantage all such Spirits as they are thus permitted to import or purchase.

8th. To receive from Government Herds 80 oxen fit for slaughter, weighing not less than lbs.¹ each, in the following proportions, viz.: 30 on 1st April 1811, 30 on 1st April 1812, and 20 on 1st April 1813.

9th. That as His Excellency the Governor has granted permission for 67,000 Gallons of Spirits to be imported into this Colony—The Contractors to have at their option the liberty to purchase not less than 10,000 Gallons⁽³⁾ of such spirits upon its arrival in part of the 45,000 Gallons.

10th. That these propositions are submitted to His Excellency in the confidence that during the Completion of the Building no further permission will be granted for the importation of Spirits⁽⁴⁾ otherwise than such quantities as the Contractors may themselves import: and should vessels accidentally arrive with Spirits on board, the Contractors to have the privilege of purchasing the same in part of the 45,000 Gallons, after the wants of Government are supplied, and such proportions granted to the Officers⁽⁵⁾ for their domestic purposes as His Excellency may deem meet.

S. BLAXCELL.
ALEX. RILEY.

Sydney,

16 Augt. 1810.

⁽¹⁾ D. Wentworth, Esq.

⁽²⁾ To be finished on or before 31st Decr. 1813.³

⁽³⁾ To be 31st Decr 1813 inclusive only.³

⁽⁴⁾ Providing those persons who have recd. such permission chuse to sell it them, but not to be compelled to do so.

⁽⁵⁾ Excepting only what Government may find necessary to import for its own use and purposes.

⁽⁶⁾ Civil and Military Publicans and Free Settlers as per schedule thereof herunto annexed.

LACHLAN MACQUARIE.

Correspondence.

USE AND MISUSE OF ANTIBIOTICS.

SIR: The question of misuse of antibiotics is an acute but a controversial one. As far as general practice is concerned, I feel, we have to answer and balance two questions before deciding on this therapy: (i) Which is the quickest and most effective way of restoring health and capacity to work in this patient? (ii) Which probably effective treatment can I withhold from this patient without delaying or endangering his recovery and ability to resume work?

¹ Gap in the original; in the contract this is stated to be 450 pounds.

³ These dates were again altered in the actual contract.

Unfortunately we are in a large number of cases not confronted with specific diseases, but with "bronchitis", "common cold", "influenza", "pyrexia of unknown origin", "abdominal colic", "fibrositis", and must decide on treatment on such "evidence", because we will cure our patients as a rule within four or five or even three days. To illustrate to the scientist how difficult it is to decide whether or not we can cure the patient quickly without antibiotics, a few typical cases may be quoted.

1. A seventy-eight-year-old lady has been under treatment for hypertension and congestive cardiac failure for years. She has a chronic cough and chronic indigestion all the time. But this time it is different. Her cough is worse, she is shorter of breath than usual, feels very weak and has a fever of 101° F. There is more bronchitis than usual and a questionable dullness over both posterior bases. Her breath has become fetid. She is "against injections". Her relatives would rather let her die "in peace". (This may become a pneumonia overnight.) Use or misuse of antibiotics?

2. A middle-aged man working in a hotel has a sore throat since a few days, which did not improve over the week-end, when he has been resting in bed, gargling and taking sulphonamide tablets, advised and issued by a chemist. His wife is just beginning to feel sore in the throat. His children are not yet affected. On examination he has a follicular tonsillitis and a "bad" breath, smelling like a streptococcal infection. His temperature is 101° F., and he feels quite sick. (A slight disease, yes. If no penicillin is given, he will infect scores of other people and feel sick quite a while, lowering his general resistance.) Use or misuse of antibiotics?

3. A girl of eighteen, whose paid sick leave has been used up earlier in the year, gets a sudden colicky pain in the gall-bladder region with persistent cough irritation, and on examination a tender, just palpable gall-bladder. No definite enlargement or downward shift of the liver. She wants to get better as fast as possible and resume work, not to lose pay. Apart from other treatment: sulphonamides or not? penicillin or not? both or not?

4. A twenty-three-year-old woodcutter appears, covered with "sores". He used to treat them with "M & B" tablets, procured from a chemist, and this has been "always effective", but this time it is not. Had to stop work. On inspection: furunculosis and impetigo. As known by the doctor from inspection of locality and previous cases, this rash is due to unhygienic living conditions in the bush, in tents and drinking water from the river. These conditions are unalterable under the circumstances. Also if this man is cured, he will come back with the same rash within foreseeable future. However, the long-standing condition has somewhat reduced his general health. He is not "very sick" of course. Use or misuse of antibiotics?

5. The doctor has been called 18 miles into the bush. It has been raining, and while stuck a few times he notices that he will probably not be able to travel the track again in the next few days, because the rain makes the road unmanageable. The child looks sick and has a temperature of 102.5° F. No other abnormality detectable. Next hospital very far off. He leaves a few drugs behind, to be used on his order only. However, nobody in the homestead is able to take a temperature or give an injection. He wants to be telephoned next day. Next day he finds out that the lines have broken down. After four days he gets a call through. The mother reports that the child had been getting worse the next day, battling for breath and wanting to cough, but could not cough properly. The mother has given him on own risk "that yellah chocolate powdah" ("Aureomycin Spersoids" in a yellow box) one teaspoonful four hourly, stuck otherwise to the general nursing directions given, and the child has improved dramatically since and coughs loosely now, apparently no fever. She took the powder because a "Macquarie Street specialist" had given it once to a baby of her sister-in-law in Sydney "straight away" on a similar occasion.

6. A blast furnace worker of twenty-two years of age is suffering from widespread impetigo since weeks, apparently infected with a number of fellow workers by a recently hired new staff member, who brought the disease from outside, but is not worried about the complaint himself. He has had 1,000,000 units of procaine penicillin in oil followed by the same amount forty-eight hours later. Both injections, supported by a course of a mixed sulphonamide, had no noticeable effect (is this a fair trial?). Tetracycline was given in 250 milligramme doses six-hourly for 16 doses, and the very disagreeable rash has been wiped out completely. Next week-end five other members of the blast furnace crew, some with affected family members, appear in the doctor's surgery, triumphantly led by the first cured

worker. Mass distribution of tetracycline? (Same thing applies to certain—not common—types of tonsillitis.)

7. An epidemic of "Influenza" is raging in the town. Average time of disability is ten days, ranging from two or three days to three weeks. During the serous catarrhal stage no doctor is called as a rule, the patient trying to cure himself with "A.P.C." (up to three tablets two-hourly). After a few days the fever is still up, and sputum and discharge become purulent and smelly. It is in this stage that the doctor is mostly called, and his impression has been that recovery is significantly speeded by administration of penicillin and "sulpha" drugs, apart from vitamins and general measures. Just this day he happens to read an article about "misuse of antibiotics" and fails to apply the treatment in the next case he gets. A day further on this very patient has developed a fully fledged pneumonia. He rushes antibiotics now, but the man is out of work for a further three weeks. Use or misuse?

These cases are only a few examples of thousands of similar kind, as they present themselves each day to thousands of general practitioners. Their very number is significant. Unfortunately bacteriological investigation is not practicable in these cases, especially in the country. As long as we have no simple bedside test to determine the sensitivity group of a patient within a few minutes or say one or two hours, we have just got to balance the dangers of delayed treatment, prolonged incapacity for work (loss of production) and spreading of infection against the cost and dangers of antibiotic injections or capsules. As our methods are crude, we are bound to err both ways from the mid-line in a number of cases. The sweeping statement "not to use" them in "slight cases" will not solve the dilemma.

The problem, however, is there. It is definitely worth while pondering about it.

Yours, etc.,

Tea Gardens,
New South Wales,
January 29, 1955.

HANNS PACY.

SIR: May I be allowed to support R. Munro Ford's unsuccessful plea for sanity and honesty in prescribing?

I am continually amazed whilst seeing children in a medical out-patient department to find certain private practitioners prescribing the mycin group of antibiotics for uncomplicated URTI's—I presume they are uncomplicated, since often there is no attempt to confine the child to the house, let alone bed, and in many cases no follow-up visit was attempted.

Certainly for the sick nervous child who is perhaps allergic to "sulpha", prone to otitis or bronchopneumonia and thunderously rebellious to penicillin one feels happier to perjure one's soul over Regulation 14A.

Again—a delightful remark at morning tea by a paragon of medical virtue: "I never use penicillin any more. Tetracycline cures everything."

Since it is obvious that these bugblasting physicians have no honesty, and pathetic appeals for common sense are no counter to the multitudinous, multithued advertisements, let us have some action to curb the law-breakers. Initially I suggest, where it is proven that extravagant prescribing has been consistently practised by any medical practitioner, then that practitioner should be compelled to pay the whole cost of his 14A prescriptions for the previous three months.

Yours, etc.,

PAMPHLET—BUT NOT DRUG-RESISTANT.

SOCIAL PERSPECTIVES IN MEDICINE.

SIR: In his address on "Social Perspectives in Medicine", Eric G. Saint gives a thoughtful account on the "shape of things to come" in medical practice. He deplores hiatuses that occur, "the perpetuation of which is not in the best interests of science or humanity, in the viewpoints of those who practise clinical medicine and surgery and those who study the many aspects of human behaviour". Dr. Saint stresses the fact that it has always been "the function of medicine to ensure that each individual is enabled to use his or her mental and physical capacity to the full, so that he or she may acquire a state of physical and mental well-being".

Throughout his article Dr. Saint points to various hiatuses in our medical services, and they are many, amongst

them being the amount of wasted "energy and research on the purely physical aspects of disease within the four walls of immense and lavishly equipped modern hospitals". Dr. Saint points out that the "needs of society will be most satisfactorily met only if we can create a more delicately poised structure of high class general practice, inspired preventive health services, intelligently operated preventive mental hygiene machinery and enlightened rehabilitative after-care for the physically and mentally disabled".

A survey of our facilities for public health in Australia shows that they are not only deficient in many respects but are incoordinated. Some compare unfavourably with most of other countries; for example, Dr. Querido, Professor of Social Medicine in the University of Amsterdam, at a lecture in London last October, spoke of "domiciliary psychiatry" in Amsterdam. This city of nearly 900,000 people under Dr. Querido's guidance overcame a shortage of psychiatrists. This shortage had been aggravated by over-hospitalization of patients with psychiatric illness on the one hand and, on the other, the unwillingness or inexperience or unpreparedness of many general practitioners to handle such patients. Now, in Amsterdam, there is a specialist domiciliary service provided on a regional basis with ten psychiatrists and twenty social workers. It gives a twenty-four hour a day service and is available at the request of a general practitioner, the police or the clergy or patients, and is centred on the University Hospital Department of Psychiatry.

In your leading article on January 29 on the long-term and the chronically ill patient you told of the needs of these unfortunate people. There is a very great need to mobilize and coordinate all the forces available—general practitioners, specialists, public health medical officers and ancillary workers—if medicine is to meet the challenge that these people present.

As it is thirty years since the Federal Royal Commission on Health made its report, many social and other changes have taken place. Has not the time come when the medical profession should recommend the holding of a second Royal Commission, not only to review the findings of the first, but to tackle new problems such as those Dr. Saint has spoken of? Such a Royal Commission should also make a review of medical education, which in many ways has not altered very much since Edwardian days. We have overloaded the undergraduate curriculum, and many of its features are not to be commended. We need a curriculum to meet the needs of our society today and tomorrow.

Yours, etc.,

E. S. MEYERS,
A./Professor of Social and
Tropical Medicine.

University of Queensland Medical School,
Brisbane,
February 15, 1955.

Medical Practice.

NATIONAL HEALTH ACT, 1953, PART IV.—PENSIONER MEDICAL SERVICE.

THE following notice appeared in the *Commonwealth of Australia Gazette*, Number 8, of February 17, 1955.

REPRIMAND OF MEDICAL PRACTITIONER.

I, Earle Page, the Minister of State for Health, hereby give notice, in pursuance of sub-section (1) of section 36 of the *National Health Act, 1953*, that I have this day reprimanded Noel Louis Fox, of Broken Bay Road, Ettalong, medical practitioner, following investigation and report by the Medical Services Committee of Inquiry for the State of New South Wales established under section 110 of the Act concerning the conduct of the aforesaid medical practitioner in relation to his provision of medical services for pensioners and their dependants under Part IV of the Act or under an arrangement made by the Director-General of Health under section 7 of the *National Health Service Act, 1948-1949*, and the *National Health (Medical Services to Pensioners) Regulations*.

Dated this 22nd day of January, 1955.

EARLE PAGE,
Minister of State for Health.

Congresses.

INTERNATIONAL SOCIETY FOR THE STUDY OF BIOLOGICAL RHYTHM.

THE fifth conference of the International Society for the Study of Biological Rhythm is to be held from September 15 to 17, 1955, at the Department of Anatomy at Karolinska Institutet in Stockholm. The principal themes are: (i) rhythmical phenomena in the nervous system, (ii) cybernetics and (iii) rhythm and industrial medicine. Applications from those who wish to read papers should be submitted prior to March 15, 1955, to Professor Ture Petrn, Karolinska Institutet, Stockholm 60, Sweden.

The Royal Australasian College of Physicians.

MEETING OF VICTORIAN FELLOWS AND MEMBERS.

A MEETING of Victorian Fellows and Members of The Royal Australasian College of Physicians will be held at the Orthopaedic Section of the Royal Children's Hospital, Frankston, on Saturday, March 26, 1955. The programme is as follows: 11 a.m., "Electromyographic Assessment of Muscle Function", Dr. D. J. Dewhurst; 11.30 a.m., "Some Aspects of Myasthenia", Dr. A. C. Schweiger; 12 noon, "Myopathies", Dr. J. Game; 12.30 p.m., lunch; 1.30 p.m., "The Painful Arm", Mr. K. C. Bradley; 2 p.m., "Osteoporosis", Dr. D. Galbraith; 2.30 p.m., "Equalization of Leg Length following Poliomyelitis", Dr. J. B. Colquhoun; 3 p.m., "Local Tetanus as a Diagnostic Problem", Dr. L. B. Cox. All members of the British Medical Association are invited to attend.

Medical Societies.

AUSTRALIAN SOCIETY OF PSYCHO-ANALYSTS.

THE Australian Society of Psycho-Analysts, which is a branch of the British Psycho-Analytical Society, held its annual interstate conference at Adelaide from January 11 to 15, 1955. At the opening meeting the Chairman, Dr. C. L. Geroe, and the Secretary, Dr. F. W. Graham, both reported on the activities of the Society for the year just passed. The members then elected for 1955 Dr. R. C. Winn as Chairman and Dr. A. Peto as Secretary. The following papers were read: Dr. C. L. Geroe, of Melbourne, "Development and Function of the Ego"; Dr. M. Hall, of Sydney, "Homosexuality and Schizophrenia"; Dr. A. Peto, of Sydney, "A Theory of Play"; Dr. R. Rothfield, of Melbourne, "Problems of Technique"; Dr. H. Southwood, of Adelaide, "Conversion Hysteria"; Dr. R. C. Winn, of Sydney, "Some Aspects of Manic-Depression"; Mrs. J. Nield, M.A., of Sydney, "Psychoanalysis of an Adolescent Girl". Dr. F. W. Graham, of Melbourne, and Dr. I. Bennet, Ph.D., of Perth, contributed to the discussions.

Notice.

UROLOGICAL SOCIETY OF AUSTRALASIA.

Open Meeting.

As part of the annual meeting of the Urological Society of Australasia, an open meeting will be held at the Royal Australasian College of Surgeons, Spring Street, Melbourne, on Tuesday, March 8, 1955, at 8 p.m. Dr. D. Stevens will speak on "Radio-active Isotopes: Can They be Usefully Applied?", and Dr. R. Kaye Scott will open the discussion. All members of the British Medical Association are invited.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED FEBRUARY 12, 1955.¹

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory. ²	Australian Capital Territory.	Australia. ³
Acute Rheumatism	2	3(3)	5(3)	..	2(1)	12
Amoebiasis
Ancylostomiasis	1	1
Anthrax
Bilharziasis
Brucellosis
Cholera
Chorea (St. Vitus)	2(2)	2
Dengue
Diarrhoea (Infantile)	4(3)	16(15)	5(2)	..	3	28
Diphtheria	10(9)	8(8)	18
Dysentery (Bacillary)	1	8(8)	1	12(1)	22
Encephalitis	2	3(3)	5
Filariasis
Homologous Serum Jaundice
Hydatid	1	1
Infective Hepatitis	78(30)	72(53)	4(3)	154
Lead Poisoning
Leprosy	2	2
Leptospirosis
Malaria	1(1)	1
Meningococcal Infection	3(1)	..	1	1(1)	5
Ophthalmia
Ornithosis
Paratyphoid	1	1
Plague
Poliomyelitis	11(8)	12(8)	8(5)	3(1)	34
Puerperal Fever
Rubella	29(24)	..	2	4(3)	1	36
Salmonella Infection	5(2)	5
Scarlet Fever	4(3)	14(10)	8	1	27
Smallpox	1	1	2
Tetanus	13(3)	13
Trachoma
Trichinosis
Tuberculosis	45(33)	11(5)	11(1)	5(4)	4(4)	3(1)	79
Typhoid Fever	1(1)	1	2
Typhus (Flea, Mite- and Tick-borne)
Typhus (Louse-borne)
Yellow Fever

¹ Figures in parentheses are those for the metropolitan area.

² Figures not available.

³ Figures incomplete owing to absence of returns from Northern Territory.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Hehir, John Desmond, M.B., B.S., 1954 (Univ. Sydney), Mater Misericordiae Hospital, Waratah, New South Wales.

Martin, James Matthew, M.B., B.S., 1945 (Univ. Sydney), Box 26, P.O., Wellington, New South Wales.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Blower, Charles Russell, provisionally registered, M.B., B.S., 1955 (Univ. Sydney); Bodlander, Fedor Max Serge, provisionally registered, M.B., B.S., 1955 (Univ. Sydney); Davey, Ronald Bruce, provisionally registered, M.B., B.S., 1955 (Univ. Sydney); Elliott, Graeme Robert, provisionally registered, M.B., B.S., 1955 (Univ. Sydney); Elphick, Richard Lansdell, provisionally registered, 1955 (Univ. Sydney); Holcome, Maureena Ena, provisionally registered, M.B., B.S., 1955 (Univ. Sydney); Hughes, Leslie Ernest, provisionally registered, M.B., B.S., 1955 (Univ. Sydney); Gallagher, Vera, provisionally registered, M.B., B.S., 1955 (Univ. Sydney); Gallagher, William Terence, provisionally registered, M.B., B.S., 1955 (Univ. Sydney); Hudson, David Lindsay, provisionally registered, M.B., B.S., 1955 (Univ. Sydney); Kan, Henry Tai-Wan, provisionally registered, M.B., B.S., 1955 (Univ. Sydney); Leckie, Thomson David, provisionally registered, M.B., B.S., 1955 (Univ. Sydney); Nossal, Gustav Joseph Victor, provisionally registered, M.B., B.S., 1955 (Univ. Sydney); Pearce, John Warren Boswell, provisionally registered, M.B., B.S., 1955 (Univ. Sydney); Quigley, Donald James, provisionally registered, M.B., B.S., 1955 (Univ. Sydney); Wald, Marx, provisionally registered, M.B., B.S., 1955 (Univ. Sydney); Woods, William Edward, provisionally registered, M.B., B.S., 1955 (Univ. Sydney); English, Cecil Hampshire, M.B., B.S., 1954 (Univ. Sydney); Georgeson, Laurence Edwin, M.B., B.S., 1954 (Univ. Sydney); Keogh, Herbert John, M.B., B.S., 1954 (Univ. Sydney); Thorburn, Norman Victor, M.B., B.S., 1954 (Univ. Sydney); Ahern, Thomas Francis, M.B., 1940 (Univ. Sydney); Buchhorn, Francis Joseph, M.B., 1948 (Univ. Sydney); Cashman, John Daniel, M.B., B.S., 1953 (Univ. Sydney); Graham, Philip Alastair, M.B., B.S., 1953 (Univ. Sydney); Kearley, Alan Spence, M.B., B.S., 1951 (Univ. Sydney); Huzella, Louis George, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act, 1938-1953*; Kocsard, Emery, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act, 1938-1953*; Stefan, Stefan George, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act, 1938-1953*.

Deaths.

THE following deaths have been announced:

INGLIS.—Edgar Montgomery Herbert Inglis, on February 13, 1955, at Balwyn, Victoria.

HART.—Albert Hughie Hart, on February 17, 1955, at Queanbeyan, New South Wales.

Medical Appointments.

Dr. N. McConaghy has been appointed senior medical officer in the Mental Hygiene Branch of the Department of Health, Victoria.

Dr. C. Bowman has been appointed a public vaccinator to the City of Mildura, Victoria.

Dr. J. A. Earl has been appointed medical registrar at the Royal Adelaide Hospital.

Dr. D. G. Stanbury has been appointed registrar at the Queen Elizabeth Hospital, Adelaide.

Dr. F. P. Callaghan has been appointed medical officer in the Mental Hygiene Branch of the Department of Health of Victoria.

Dr. E. R. White has been appointed a member of the Medical Board of Victoria.

Dr. P. T. J. C. P. Warner has been appointed Medical Research Fellow in the Institute of Medical and Veterinary Science, Adelaide.

Dr. H. J. Prior has been appointed medical superintendent in the Division of Mental Hygiene, Department of Public Health, New South Wales.

Dr. J. L. Evans has been appointed deputy medical superintendent in the Division of Mental Hygiene, Department of Public Health, New South Wales.

Dr. A. Fryberg has been appointed chairman of the Nurses and Masseurs Registration Board of Queensland.

Diary for the Month.

- MARCH 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
- MARCH 11.—Tasmanian Branch, B.M.A.: Branch Council.
- MARCH 11.—Queensland Branch, B.M.A.: Council Meeting.
- MARCH 12.—Tasmanian Branch, B.M.A.: Annual Meeting.
- MARCH 14-18.—New South Wales Branch, B.M.A.: FOCLA Post-Graduate Course at Wollongong.
- MARCH 14.—Victorian Branch, B.M.A.: Finance Subcommittee.
- MARCH 15.—New South Wales Branch, B.M.A.: Ethics Committee.
- MARCH 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.
- MARCH 16.—Victorian Branch, B.M.A.: Clinical Meeting.
- MARCH 17.—Victorian Branch, B.M.A.: Executive of Branch Council.
- MARCH 19.—Western Australian Branch, B.M.A.: Annual General Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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